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Name of Candidate: Kristy L Morris
Master of Science
Department of Medical and Clinical Psychology
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Thesis and Abstract Approved:

_____ _____

Tracy Sbrocco, Ph.D. Date
Department of Medical and Clinical Psychology
Committee Chairperson

_____ _____

Kelly Rohan, Ph.D Date
Department of Medical and Clinical Psychology
Committee Member

_____ _____

Michele Carter, Ph.D Date
Department of Psychology, American University
Committee Member

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Traditional obesity treatments have demonstrated successful short-term, but not long-term, weight loss. Behavior Therapies (BT) typically prescribe moderate kilojoule reduction and eating rules that promote a "dieting mentality." Behavioral Choice Treatment (BCT), which emphasizes mild kilojoule reduction and flexibility in eating has previously demonstrated greater weight loss maintenance compared to BT. This study compares eating patterns across treatments to explore differences related to weight loss. Thirty-two obese women, randomly assigned to BT or BCT, used computerized eating diaries to record all foods consumed. At treatment onset, both groups reduced kilojoule (BT, p<.01; BCT, p<.01) and percent fat (BT, p<.01; BCT, p<.01) intake. At the end of treatment, however, BCT reported greater kilojoule [p<0.05] and percent fat [p<0.01] intake. No group differences were found for meal frequency or variability in kilojoule intake. These data suggest slight rather than moderate caloric restriction and greater flexibility in eating may promote the observed long-term weight management in BCT over BT.

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Kristy Lauren Morris
Department of Medical and Clinical Psychology
Uniformed Services University of the Health Sciences

ABSTRACT

Title of Thesis: "A Comparison of Eating Patterns Across Two Obesity Treatments: Behavior Therapy vs. Behavioral Choice Treatment"

Author: Kristy Lauren Morris, Masters of Arts, 2003

Thesis directed by: Tracy Sbrocco, Ph.D.

Associate Professor

Department of Medical and Clinical Psychology

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A COMPARISON OF EATING PATTERNS ACROSS TWO OBESITY TREATMENTS: BEHAVIOR THERAPY VS. BEHAVIORAL CHOICE TREATMENT

by

Kristy Lauren Morris

Master's Thesis submitted to the Faulty of the Department of Medical and Clinical Psychology Graduate Program of the Uniformed Services University of the Health Sciences in partial fulfillment of the requirements for the degree of Master of Science, 2003

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could not have happened.
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Epidemic of Obesity and Overweight in the US

It is estimated that over 97 million Americans are overweight or obese (NHLBI, 2000). According to the third National Health Survey (US Department of Health and Human Services; USDHHS, 1994), the combined prevalence for overweight and obesity in the United States was approximately 59% for men and 51% for women. More recently, a 1999-2000 survey indicated a further increase in overweight, from 59% to 64.5% (Flegal, Carroll, Ogden, & Johnson, 2002). The prevalence for overweight and obesity is even greater for minority women, found in 66% of African American women and 66% of Hispanic women compared to 49% for Caucasian women (Flegal, Carrol, Kuczmarski, & Johnson, 1997). From 1960 to 1994, overweight has increased roughly 2% and obesity has almost doubled, increasing from 13% to now 26% (USDHHS, 2003). This trend has been so dramatic, the Healthy People 2010 publication (USDHHS, 2000) declared the reduction of overweight and obesity a national health objective and the Surgeon General (USDDHS, 2001) has made a call to action for its prevention and treatment.

The Health Consequences of Obesity and Overweight

Today, overweight and obesity have been labeled as the second leading cause of preventable death in the United States, attributed to an estimated 300,000 deaths per year (National Heart, Lung, & Blood Institute; NHLBI, 2000). Because of the associated physiological, economic/social, and psychological consequences, overweight and obesity are now major public health concerns. The National Heart, Lung, and Blood Institute (NHLBI) Obesity Initiative Task Force (1998) defines overweight as a body mass index (BMI) of 25 to 29.9 kg/m^2 and obesity as a BMI of 30 kg/m^2 and above. Conceptually, a

body mass of 30 is equivalent to a woman who is 5'6" and weighs approximately 180 lbs., and to a man who is 6'9" and weighs about 205 lbs. These criteria were developed based on epidemiological data showing an increased risk of morbidity and mortality associated with body mass indexes above 25 kg/m^2 (NHLBI, 2000).

According to the National Institutes of Health (NIH) Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults (1998), individuals with a BMI of 25kg/m^2 or above are at greater risk for premature death as a consequence of overweight and obesity. These risks increase as an individual's BMI increases and the prevalence of increased risk is consistent across racial and ethnic groups for all health conditions (Flegal et al., 1997). It has been found that weight loss as small as 5%-10% of initial weight is associated with a reduction in blood pressure, serum triglycerides, total cholesterol and LDL cholesterol, and blood glucose, and an increase in HDL cholesterol (Devlin, Yanovski, & Wilson, 2000). This suggests that weight loss and weight loss maintenance are important in the prevention and reduction of illnesses related to overweight and obesity.

Physiological Consequences. Extensive research has provided strong evidence concerning the effects of overweight and obesity on physiological health. Excess body fat has been associated with the onset of Type 2 diabetes, increased risk for hypertension, coronary heart disease, respiratory disease, and increased levels of triglycerides and decreased high-density lipoprotein, both of which are recognized as risk factors for cardiovascular disease (Pi-Sunyer, 2002). Overweight and obesity have been associated with certain forms of cancer such as colon, prostate, uterine, and ovarian cancer (NIH, 1998), as well as sleep apnea, osteoarthritis, gynecological problems, and gallstones

(NHLBI, 1998). There has also been evidence that obesity is associated with an increase in mortality rate of 50%-100% compared to normal weight individuals (NIH, 1998).

Social/Economic Consequences. Obesity is noted as one of the most stigmatized physical attributes in Western Society (Higgs, et al., 1996). Persons who are obese face a number of situations where they experience social discrimination, including decreased perception of attractiveness, reduced employment opportunity (Allon, 1982), and reduced social status (Foreyt, Walker, Poston, & Goodrick, 1996). As Brownell (1982) noted, obese persons are not only stigmatized regarding their weight, they are also considered responsible for it.

The health consequences have had a tremendous impact on the economic costs of obesity. In 1995, it was estimated that the total cost attributable to obesity in the US was $99.2 billion in both direct and indirect costs. Direct costs, including prevention and treatment, totaled $51.6 billion. Indirect costs, including lost wages due to illness or disability, was $47.6 billion (Wolf & Colditz, 1998).

Psychological Consequences.

Little conclusive evidence is available regarding the psychological consequences of obesity. Whereas some studies have demonstrated that obesity is potentially associated with increased psychopathology, particularly anxiety disorders, depression, substance abuse, and increased body dissatisfaction (Fitzgibbon, Stolley, & Kirschenbaum, 1993; Higgs, et al., 1996; Lapidus, Bengtsson, Hallstrom, & Bjorntorp, 1989; Prather & Williamson, 1988; Sullivan et al., 1993), other studies have found no such differences (O'Neil & Jarrell, 1992; Striegel-Moore & Rodin, 1986; Stunkard & Wadden, 1992; Wadden & Stunkard, 1985). It has been consistently shown that obesity

treatment seekers are more likely than non treatment seekers or normal weight controls to exhibit greater psychopathology; up to 5% meet criteria for at least one DSM-III-R (American Psychiatric Association, 1987) disorder (Higgs, et al, 1996).

Evidence suggests cultural differences exist regarding the psychological consequences of overweight and obesity. In spite of their generally higher weight status, African American women experience less social pressure about weight loss and thinness compared to Caucasian women (Striegel-Moore, et al., 1996). Also, regardless of weight status, African American women consider themselves more attractive than Caucasian women (Kumanyika & Keil, 1994).

The association of obesity and depression is now the focus of newer research. Evidence from studies using questionnaire methods that have examined the temporal relationship between obesity and depression has been mixed, as some have found obese individuals are at greater risk for depression (Istvan, Zavela, & Weidner, 1992; Ross, 1994), some have found the inverse to be true (Crisp, 2000; Palinkas, Wingard, Barrett-Conner, 1996), and others found no association (Friedman & Brownell, 1995). More recently, Roberts, Deleger, Strawbridge, and Kaplan (2003) used DSM-IV symptom criteria for major depressive episode and found that obesity is associated with an increase risk of depression over time. This link is particularly true for women (Goodman & Whitaker, 2002).

Dieting and Obesity Treatment

As the prevalence of obesity has continued to rise, so have efforts for weight loss, most often in the form of dieting. It has been noted that as many as 75% of women and 47% of men in the U.S. reported having dieted at least once in their lifetime. In 1990,

approximately 23% of men and 40% of women were actively trying to lose weight (Jeffery, Adlis, & Foster, 1991). Interestingly, it has also been found that, in spite of the greater prevalence of overweight and obesity in minority populations, an estimated 83% of those dieting were Caucasian, compared to 9% African American and 7% Hispanic (Horm & Anderson, 1993). This discrepancy between weight and dieting is most likely explained by cultural differences in perception and acceptance of weight and body image satisfaction. Drive for thinness appears normative among Caucasian girls and women, even among those normal weight (Fallon & Rozin, 1985; Klesges, 1983; Garner, Garfinkel, Schwartz, & Thompson, 1980), whereas African American girls and women perceive themselves as less overweight and have greater body acceptance than Caucasians (Kumanyika, 1994; Miller, et al. 1988; Story, French, Resnick, & Blum, 1995).

Historical Perspective of Obesity Treatments

As efforts for weight loss have increased, so too have efforts in the development of successful weight loss treatments. Historically, obesity was seen as a problem of overeating. The basic assumption was that calorie intake greater than calorie expenditure would result in weight gain. The focus of treatment, therefore, was simply caloric restriction. Results of treatments following this principle, however, were disappointing. Stunkard (1958), in a review of weight loss treatments, summarized his findings as follows:

> "Most obese persons will not stay in treatment for obesity. Of those who stay in treatment, most will not lose weight, and of those who do lose weight, most will regain it" (p. 79).

Behavior therapy was introduced in weight loss treatments to overcome the lack of effectiveness in caloric restriction alone. The focus remained on food consumption, but there was a shift in emphasis from simply overeating to faulty eating habits (Wooley, Wooley, & Dyrenforth, 1979). Attention turned to situational variables associated with eating, including both external and emotional cues. It was thought that overweight persons had a tendency to over-respond to external cues and under respond to internal cues (Schachter & Rodin, 1974), and therefore there was an acknowledgement of the influence of times, social context (people and places), and emotions in eating. This led to the implementation of behavioral techniques in an attempt to recognize and modify such influences in order to improve overall weight loss. The goal of treatment, then, was to promote weight loss by using behavioral techniques to eliminate eating behaviors that reduce caloric intake and contribute to excess food consumption and weight gain.

Basic strategies in the behavioral treatment for weight loss have been self-monitoring, stimulus control, modifying eating style, and reinforcement (Perri & Fuller, 1995). Self-monitoring is the systematic observation and recording of target behaviors (Kanfer, 1970), used in weight loss treatment programs to identify faulty eating behaviors and to target areas of change. Self-monitoring gives the individual the opportunity to recognize and evaluate their eating behavior by providing a vehicle for self awareness (Heatherton & Baumeister, 1991) and gives researchers or clinicians the opportunity to

learn more about the components that led to the development and maintenance of weight gain.

Stimulus control is the attempt to break the leaned associations between environmental cues (situations or events) and problem eating behaviors (Foreyt & Goodrick, 1993). Popular stimulus control techniques used in conventional weight loss therapy include ridding the household of "forbidden foods" in order to eliminate temptation, making certain environments off limits to eating, eating only in “eating places,” and restricting meals and snacks to specific times during the day. For example, stimulus control would make environments such as in front of the television or in the car off limits during eating because they divert the individual’s attention away from eating. Examples of modification of eating style include slowing the rate of eating and putting utensils down between bites of food. Reinforcement techniques in obesity treatment set reward contingencies for treatment attendance, adherence, or reaching certain weight loss goals.

Behavior modification techniques, together with caloric restriction, have been the conventional method used in weight loss treatments for over 2 decades (Perri & Fuller, 1995). Overall, experts agree on the short-term success of behavior therapy on weight loss (NHLBI, 2000). In a review of treatments, Wadden (1993) noted that the amount of weight loss associated with behavior therapy doubled from that of earlier treatments. More recently, the NIH’s (1998) report concluded that the combination of caloric restriction with behavior modification produces greater weight loss compared to dietary treatments alone. Despite the significant improvements in weight loss, however, these

losses were short-term with most of the weight lost during treatment regained within 2 years of follow up (Brownell, 1982; Perri, 1998).

At this time, weight maintenance remains the exception in overweight and obesity treatment. Until the recent stepped care approach proposed by the National Heart, Lung and Blood Institute and the North American Association for the Study of Obesity panel (NHLBI & NASSO; 2000), little has been done for individualized obesity treatments. This is curious because ideographic tailoring of treatment has been advocated for almost 2 decades (Brownell & Wadden, 1991). In order to tailor treatment, we must understand what treatment, or what specific aspects of treatment, works for which individuals. The weight loss registry of successful maintainers is an example of this approach; these data show that traditional behavior strategies do not work for the larger segment of the population (Jeffery et al., 2000).

Little, however, has been done to understand why certain patterns of self-regulation work in the short-term, but fail during maintenance, or why certain stimulus response methods do not work for a large segment of the population. A better understanding of the mechanisms behind treatment effects would enable researchers and clinicians to improve treatment methods.

Models of Eating and Self-Regulation

The emergence of theories to explain differences in obese vs. normal weight persons' eating behaviors began roughly 30 years ago with the work of both Schachter (1968; 1971; Schachter & Rodin, 1974) and Nesbitt (1972). Schachter (1968) proposed a psychosocial theory, the externality hypothesis, which suggested that normal weight and overweight persons have different cues to signal hunger and satiety. He suggested

that normal weight people eat in response to internal physiological cues (hunger pains and feelings of fullness) whereas obese people tend to respond to external cues, such as sights and smells of food.

Schachter, Gordon, and Goldman (1968) experimentally investigated the externality hypothesis by inducing fear (either high or low threat of shock) in overweight and normal weight participants who were either food deprived or full. Results indicated that normal weight participants who were food deprived ate less when fear was high compared to when fear was low. The opposite was found for overweight participants. Overweight participants who were food deprived ate more when fear was high compared to when fear was low. This supported the hypothesis that eating is internally controlled within normal weight individuals and externally triggered in overweight individuals.

A number of subsequent studies indicated that subjective reports of hunger differ between obese and normal weight individuals with respect to food deprivation and physiological vs. external triggers for eating (Pliner, 1974). Not all studies supported this conclusion, however, nor did all researchers agree with Schachter's externality hypothesis. Nisbett (1972) proposed an alternative theory, which suggested that each individual has a biological "set point" for food consumption determined by the number of fat cells in the body. Because overweight individuals have a greater number of fat cells, they naturally have a higher set point than normal weight individuals. Societal pressures to be thin drive overweight people to restrain their eating in order to lose weight, leading them to eat below their set point. Eating below their biological set point by restraining their eating leaves obese individuals starving by biological standards, resulting in eating

behavior that is more externally controlled (Ruderman, 1986) and, therefore, making the individual more likely to over eat.

Nisbett's set point theory sparked interest in the role of dieting on eating, leading to the development of the construct of restraint (Herman & Mack, 1975; Ruderman, 1986) and later, the restraint theory (Herman & Polivy, 1975) and the boundary model of eating (Herman & Polivy,1980). Most studies initially testing Nisbett's hypothesis assumed that it was overweight people who were below their biological set point because of the pressure to lose weight by dieting compared to normal weight people, who remained within their set point. There was no evidence, however, that supported the dichotomy in restrained eating between overweight and normal weight persons (Ruderman, 1986). Herman and Polivy (1975) expanded Nisbett's idea by suggesting that normal weight individuals are also influenced by a cultural drive for thinness. This drive for thinness leads them, like overweight individuals, to restrain their eating to below their natural set point in order to lose weight, resulting in a vulnerability for externally triggered eating and overeating (Lowe, 1993). Many more individuals, therefore, regardless of their weight, are at risk for dysregulated eating.

Herman and Mack (1975) tested the hypothesis that it is restrained eaters rather than weight status that is associated with a vulnerability to external cues for eating and overeating in college-age females. Participants were placed in a 0, 1, or 2 milkshake preload condition, then asked to sample ice cream. A questionnaire was used, which later became the Restraint Scale (Herman & Polivy, 1975), to identify individuals who were concerned with their weight and who voluntarily engage in restricting their diet. High restraint, normal weight participants ate more (ice cream) after a preload compared to no

preload; low restraint subjects ate less as the size of their preload decreased. This study showed that it was not weight status – obese vs. normal weight – that influenced eating patterns as much as it was restraint over eating. Low restraint participants displayed eating patterns consistent with Nisbett's initial conceptualization of normal weight eaters, responding to internal, physiological hunger cues. Similarly, participants high in restraint, even at normal weight, ate in line with the characteristics originally conceptualized as the overweight eating patterns; restrained eaters were more likely to overeat in response to external food cues. This finding has been replicated numerous times (Herman & Polivy, 1980; Lowe, 1993; Westenhoefer, 1991).

In Herman and Mack's (1975) experiment, an important finding was that high restraint eaters ate more after the milkshake preload than they did with no preload, where low restraint eaters ate more after no milkshake preload. This "counter-regulatory effect" described restrained eaters' tendency to exhibit a dietary disinhibition after a preload (Lowe, 1993). Herman and Mack (1975) explained this counter-regulatory effect by suggesting that once restrained eaters break the eating restrictions they have placed on themselves, they come to lack any restraint at all. Herman and Polivy (1980), in their restraint theory, explained the cognitive processes involved in dietary restraint and the effects they have on eating behavior. They suggested that restrained eaters go through periods of caloric restriction (dieting) and overeating that occur when efforts to restrain eating are interrupted by some event, known as a disinhibitor.

After the restraint theory, Herman and Polivy (1984) developed the "boundary model," which integrated the restraint theory into a broader physiological and psychological/social model of eating. In the boundary model, Herman and Polivy (1984)

proposed a biological pressure to maintain food consumption within the range of hunger and satiety called the zone of "biological indifference." In this model, restrained eaters (dieters) and unrestrained eaters are differentiated. Normal eaters are thought to start to eat when they experience aversive stimuli associated with hunger and stop eating when they experience aversive stimuli associated with satiety. Restrained eaters are believed to have lower hunger boundaries and higher satiety boundaries than normal eaters. That is, it takes more food deprivation for restrained eaters to experience hunger and they need to consume more food to begin feeling "full" compared to normal eaters. Dieters are also believed to have a third boundary, the "diet boundary." The dieting boundary is the zone between hunger and satiety which operates on a cognitive rather than physiological level, particularly in terms of rules that regulate eating. Once this boundary is crossed, and rules are broken, dieters are likely to overeat or continue to eat until they reach their satiety boundary.

In the boundary model, Herman and Polivy (1984) explain the counter-regulatory eating phenomena found by Herman and Mack (1975) in their preload manipulation experiment. Restrained eaters' tendency to overeat after a preload compared to exhibiting restraint with no preload is due to a violation of their diet boundary; once a dieter crosses their diet boundary, restraint is disinhibited and eating continues until his or her satiety boundary is reached. The diet boundary, however, is not only influenced by physiological cues such as amount of calories eaten. Evidence indicates that disinhibition is conditional on the perception of overeating or crossing the diet boundary rather than the physiological response to actual amount of calories consumed (Heatherton, Polivy, & Herman, 1990). That is, it is a cognitively regulated phenomena.

This evidence supports cognitive mechanisms as having a key role in dieting and disinhibition. Dieters tend to have extremely rigid rules regarding their food intake and think of dieting as an all-or-none attempt at controlling eating. Once unrealistic dietary rules are violated, the dieter will give up all rules and overeat. After violating dietary rules, cognitions that follow may include, "I've blown it, I've ruined my diet today – I may as well just eat whatever I want, I can always start again tomorrow" (Ruderman, 1986). This process is often referred to as "dieting mentality" (Brownell & Rodin, 1994).

Dietary disinhibitors can also be environmental or emotional variables (Heatherton, Polivy, & Herman, 1990; Ruderman, 1986). Much of the research on emotional disinhibitors has been on depression and anxiety and is often referred to as emotional induced eating. Studies consistently demonstrate that emotions, particularly are more likely to affect eating behaviors in obese individuals compared to normal weight individuals. This has been found in both treatment and non-treatment seekers (Fitzgibbon, Stolley, Melinda, & Kirschenbaum, 1994; Leon & Chamberlain, 1973; Lowe & Fisher, 1983), as well as naturalistic and prospective studies (Ganley, 1989; Glucksman, Rand, Stunkard, 1978; Lowe & Fisher, 1983). Research on environmental cues has included any cue in the environment that triggers food cravings. Because of individual differences, specific environmental cues are widely variable.

Current Treatment Research Outcome

Over the past 2 decades, researchers have put forth great efforts in an attempt to overcome the recidivism in weight loss treatments (NHLBI, 1998). In the 1980's and 1990's, a surge of treatments emerged with an expanded treatment focus, including

decreasing the level of caloric restriction, increasing treatment length, and comparing different and multiple combinations of treatment components. The goal was to find a treatment that promoted significant weight loss and maintained that weight loss over time.

Standard behavioral treatments for obesity commonly prescribe a moderate caloric restriction, prescribing an intake of 1000 - 1200 kcal/day (NHLBI, 2000; Perri, Nezu, Patti, & McCann, 1989) as well as certain standard methods for weight loss. "Banking calories" is a behavioral strategy used where clients are encouraged to restrict calories if they have eaten or anticipate eating over the recommended caloric limit. Evidence has shown that moderately restricting caloric intake to approximately 1200 kcal/day leads to approximately an 8.5 kg in 20 weeks (Wadden, 1993), however 95% of people regain weight lost during treatment over 2-5 years of follow up (Perri, 1992).

Consistent weight regain after treatment led researchers to investigate whether diets with a greater level of caloric restriction than the standard 1200 kcal/day would lead to and maintain initial losses (Wadden, 1993). Very-low-calorie diets (VLCD), which typically include a daily intake of 400 to 800 calories plus vitamin and mineral supplements (Perri & Fuller, 1995) were developed to test this. Results demonstrated significantly greater weight losses compared to conventional 1000 -1200-calorie diets. In Wadden's (1993) review of clinical research trials, the average weight loss following a VLCD was approximately 20kg over 12 to 16 weeks, which was more than double the amount of weight loss and at a faster rate than with standard treatment. At 5-year follow up, however, in both diets with standard caloric restriction and VLCDs, weights returned

to baseline. Thus, like moderately restrictive diets, VLCD's have not demonstrated clinical efficacy over the long-term.

Current behavioral treatments for obesity are between 16 and 24 weekly sessions, with some extending treatment to include biweekly or monthly contacts (Leemakers, et al., 1999). Two reviews of behavior therapy for obesity (Bennett, 1986; Perri, Nezu, Patti, & McCann, 1989) found treatment length to be the key factor in the amount of weight lost during treatment. It can be concluded that greater weight loss occurred because participants were engaging in behaviors conducive to weight loss for longer periods of time. In spite of these greater initial losses, weight regain occurred after the extended treatment ended (Wadden, Foster, & Letizia,1994). More recently, Perri and Corsica (2002) reviewed the weight loss maintenance literature and found that treatment strategies aimed at promoting the maintenance of weight loss have demonstrated modest benefits. Extending therapy length, as well as increasing physical activity, were found to have the greatest impact on improving long-term weight loss maintenance.

Even with these modifications, however, treatments have stayed within the boundaries of conventional behavioral treatments: limiting caloric intake, limiting food choices, and limiting the times for and the social context of eating. From a historical perspective, the short-term success of weight loss itself via conventional behavioral therapy is impressive (Perri & Fuller 1995; Stunkard, 1958; Wadden, 1993), however, results remain temporary (Perri, 1998). Across behavioral dieting treatments, the observed pattern of weight change has been consistent: initial weight loss is rapid then declines; weight is regained and eventually plateaus close to baseline weight (Cooper & Fairburn, 2002; Jeffery et al., 2000). Overall, it is safe to conclude that today weight

regain is still the rule rather than the exception (Sbrocco, Nedegaard, Stone, & Lewis, 1999).

Questioning the Role of Dieting

Concern among researchers regarding the lack of effectiveness of dieting and its potentially hazardous effects on physiological health and eating behaviors has grown in the scientific community. Frustration stems from the seemingly inevitable relapse rates for weight loss treatments and the increasing prevalence of eating disorders, which is thought to be related to dieting (Presnell & Stice, 2003). The role of dieting in weight loss treatment me into question and an increase in studies on dieting behavior emerged (Brownell & Rodin, 1994; Lowe, 1993). Attention turned to negative features of dieting, including the potential risks of weight cycling and the cognitive and behavioral consequences of food restriction.

Weight cycling, commonly called "yo-yo dieting," is the chronic dieter's constant cycle of dieting and overeating that results in repeated weight loss and weight regain (Lowe, 1993). Many scientists became concerned with the negative effects of weight cycling including metabolism and weight loss, health and mortality, and psychological well being. Findings to date have been mixed and/or inconclusive according to a review by the National Task Force on the Prevention and Treatment of Obesity (1994).

Brownell, Greenwood, Sellar, and Shrager (1986) launched research on the effects weight cycling on metabolism and weight by examining the effects of repeated weight loss and regain on rats. They found that weight cycled rats gained weight easier and had more difficulty losing weight than non-weight cycled rats. Weight cycling is believed to have harmful effects on health and mortality. Chronic weight cycling has

been related to renal cell cancer in women (Lindbald, Wolk, Berfstron, Persson, & Adami, 1994) and could potentially increase oxidative DNA damage, which has been identified as a biological marker of cancer risk (Uhley, Pellizzon, Buison, Guo, Djuric, & Jen, 1997). In some studies, repeated episodes of weight loss and weight regain have been associated with increased hypertension (Ascherio, Rimm, Giovannucci, et al., 1992), diabetes (Blair & Paffenbarger, 1994), increased cardiovascular disease (Blair, Shaten, Brownell, Collins, & Lissner, 1993; Lissner et al., 1991), and increased mortality (Manson, Wilett, Stampfer et al., 1995).

Research over time, however, yielded conflicting findings regarding the effects of weight cycling on health. Because of this, the National Task Force on the Prevention and Treatment of Obesity (1994) conducted a review of the weight cycling literature. It was concluded that, despite the popular acceptance of weight cycling's negative effects on metabolism and health, a careful review of studies did not support this. The Task Force concluded not only that weight cycling has no negative effects on body composition, metabolic rate, or weight loss, but also that the benefits may even outweigh the risks, as a weight loss of as little as 5 to 10 pounds has been associated with improvements in obesity-related conditions such as diabetes and high blood pressure.

Concern regarding the effects of weight cycling on psychological health has also been raised. It has been proposed that weight cycling has negative consequences on psychological health. Clinically, obese individuals report feelings of guilt, shame, and inadequacy after weight regain (Wadden & Wingate, 1995), and empirically, weight-cycling has been found to be associated with lower general well being and perception of health (Foreyt et al., 1995). In a review of the psychological effects of weight cycling,

however, Foster, Sarwer, and Wadden (1997) reported that weight cycling has not been found to be directly associated with depression, psychopathology measured by the Minnesota Multiphasic Personality Inventory-2 (MMPI-2), stress, or dysfunctional thoughts associated with mood disturbances.

This cycle of dieting and overeating can also occur in relatively brief periods without notable weight changes, otherwise called "chaotic eating" (Lowe, 1993). Chaotic eating is characteristic of individuals with dieting mentality. Again, the dieting mentality occurs when dieters have extremely rigid rules regarding their food, which, once violated, results in abandoning all rules and overeating. These maladaptive cognitions related to food restriction may not follow actual dietary restriction (Lowe, 1993). As noted above, Polivy (1976) found neither restrained nor unrestrained eaters regulated eating based on true caloric content, but rather in response to cognitions regarding caloric content. Dieting treatments may, by design, result in chaotic eating patterns, which can be seen by examining the daily caloric content of participants in traditional dieting treatments for obesity (Clinical observation, 2002). Dieting treatments promote caloric restriction to compensate for periods of overeating. This may result in brief periods of restriction, ranging from several hours to much longer periods, such as several days of highly restricted eating. Chaotic eating may contribute, in part, to the failure of weight loss maintenance.

Restraint and Overeating

Traditional behavior therapies typically employ a moderate caloric restriction of 1000 -1200 kcal/day and rules that promote abstinence from specific high calorie/high fat foods in order to promote weight loss (NHLBI, 2000). Strategies used to achieve this

include stimulus control techniques and banking calories, including meal skipping. These strategies, along with the expectations of rapid weight loss, are believed to set dieters up to fail. Over restriction of food intake and adoption of rigid rules regarding eating may set individuals up for overeating and unplanned eating episodes. Studies have indicated that both unplanned meals and overeating episodes increased the probability of subsequent episodes (Schlundt, Sbrocco & Bell, 1989). Evidence also suggests that skipping meals increases the probability of impulsive and or over eating (Johnson, Corrigan, Schlundt, & Dubbert, 1990). This phenomena is often explained by the abstinence violation effect (Cummings, Gordon, & Marlatt, 1980).

The abstinence violation effect (AVE) was first introduced in the addiction literature to explain the emotional and cognitive response to violating efforts to abstain from substance use. This model suggests that individuals who lapse after committing to abstain either make global negative attributions ("I have no willpower") or more specific attributions ("this was a high risk situation"). Those who make global attributions are likely to either feel negative affect, such as guilt, or to believe they have little control. Those that feel negative affect are more likely to further engage in the specific behavior they are trying to avoid, such as drinking or drug use, in order to cope with the affect. Those that believe they have little control are more likely to abandon abstinence all together. This effect has been suggested to occur in eating behavior. The abstinence violation effect found its place in the eating disorders literature when dieting was first functionally related to binge eating (Brownell & Rodin, 1994; Garner & Garfinkel, 1985). Evidence from both the eating disorders and obesity literatures found that overweight individuals had a tendency to binge eat (Loro & Orleans, 1981; Wilson,

1976), binge eaters had a tendency to be overweight, and dieting and binging often co-occur (Polivy & Herman, 1985). This evidence suggests some relationship between dieting and binging exists; however, a direct causal relationship has not been established.

Beyond eating disorders, there is evidence that the abstinence violation effect occurs in persons who are dieting. The same cognitive mechanisms may be operating among dieters who binge eat and dieters who overeat. In these groups, it is possible that perception of restriction is an antecedent to overeating or unplanned eating. Perhaps it is moderate caloric restriction that promotes abstinence, creating an environment where it is desirable to demonstrate high restraint, but the likelihood of violating abstinence rules is high. Once a lapse, or situation which violates abstinence rules, occurs, negative cognitive and emotional responses follow. Individuals may feel guilty about breaking dietary rules, and to cope with this guilt, they may continue to eat. Cognitions may include, "I already blew it today, I'll start again tomorrow." Individuals may also make global attributions about their lapse, such as "I blew it, I have no willpower," which, in turn, leads to subsequent episodes of overeating and unplanned eating.

More mild restriction, on the other hand, may promote greater flexibility in cognitions regarding eating behavior (i.e., dieting rules that are much more reasonable compared to those found in diets with greater restriction). More reasonable rules are not only easier to adhere to, they also make a lapse much less likely to occur because more reasonable rules are associated with greater flexibility in eating behavior compared to strict rules that promote rigid thinking. Therefore, when a lapse occurs in a treatment involving more mild restriction and more flexible rules, the individual may attribute the lapse to a specific high-risk situation, thus leading to no abstinence violation effect.

High-risk situations, such as negative emotional states and interpersonal and environmental situations (Brownell, Marlatt, Lichtenstein, & Wilson, 1986), may get in the way of maintaining caloric restriction and abstinence rules. A functional analysis of situational and environmental variables and eating behavior supported the abstinence violation effect; once one dietary slip has occurred, there is an increased probability of a subsequent slip (Johnson, Corrigan, Schlundt, & Dubbert, 1990). This implies that the restrictive nature of traditional dieting strategies sets the stage for individuals in traditional dieting treatments to fail.

Alternative Approaches

A great deal of effort has gone into identifying the etiology and maintenance of obesity and the consequences of unhealthy eating behaviors, including dieting itself and the lack of weight loss maintenance in the treatment for obesity. In response to the potential risks associated with dieting, the idea that dieting leads to binge eating, and the consistent lack of clinically significant weight loss and long-term weight loss maintenance, new approaches to weight loss have emerged, including treatments based on models of non-dieting and individualized treatments.

The idea that dieting should not be used in obesity treatment is based on cumulating evidence that diets do not work and are associated with negative physical and psychological consequences, with costs that far outweigh the benefits (Foster & McGuckin, 2002). Dieting has adverse effects on cognitive performance, body image, mood, and binge behavior and has been found to precipitate a preoccupation with food and eating (Cogan & Ernsberger, 1999). The restrictive nature of dieting may also create

a vulnerability to counter-regulatory eating (Lowe, Foster, Kerzhnerman, Swain, & Wadden, 2001), which may ultimately be a set up for weight loss failure.

Non-dieting treatments promote the abandonment of diets and acceptance of current weight. Programs differ in the methods they employ, however, all typically attempt to educate participants regarding dieting's negative effects and lack of effectiveness, challenge participants' ideal of thinness, encourage participants to abandon dieting practices such as food avoidance, teach participants to re-learn natural hunger cues, and increase self-esteem through weight acceptance rather than weight loss (National Task Force of the Prevention and Treatment of Obesity, 2000). It is believed that once acceptance of weight occurs, weight loss may subsequently occur as a result of a decrease in eating pathology and an increase in psychological health.

Several studies have evaluated the non-dieting approach. In a 10-week non-dieting pilot program, Polivy and Herman (1992) found that participants reported an increase in mood and self-esteem after treatment. Weight, however, also increased an average of 3.5 kg during the 6 months after treatment. Similar improvements in psychological functioning were found in other studies of non-dieting treatments (Ciliska, 1998; Roughan, Seddon, &Vernon-Roberts, 1990). Reports of weight gain, however, have varied. In a comparison of a cognitive therapy non-dieting program with a behavior therapy weight loss program, Tanco, Linden, and Earle (1998) found that a small amount of weight loss (1.6 kg) occurred in the non-dieting group, although no significant changes were found in psychological functioning in either group.

Overall, results for non-dieting approaches suggest an improvement in psychological well being and eating pathology, but with little weight change. This lack

of treatment effects on weight loss maintenance raises some concern about leaving obesity untreated because of its various associated health risks. Lack of weight change in non-dieting approaches does not effectively address physiological consequences of remaining obese compared to losing weight.

In addition to non-dieting treatments, there are proponents for more individualized treatments, including individualized behavioral assessment and behavioral analysis of impulsive eating and overeating in persons seeking to lose weight. The need for more individualized treatment is supported by the work of Schlundt and colleagues (1989), who used a functional analysis to examine high-risk situations leading to abstinence rule violations, over eating, or impulsive eating. Identified high-risk situations included positive social interactions, negative emotions, and physiological craving. High-risk situations were also influenced by person-specific situational variables and individual characteristics. Specific outcome data on individualized treatments are not available.

Behavioral Choice Treatment

Behavioral Choice Treatment (BCT) was developed in response the concerns with the lack of effectiveness of traditional dieting treatments. BCT is a skills training program based on a decision-making model of individuals' food choices (Sbrocco, Nedegaard, Stone, & Lewis, 1999). The decision-making model of food choices uses decision theory to link specific situational and environmental eating behaviors to eating outcome and goals (Sbrocco & Schlundt, 1998). The decision-making model suggests that eating behaviors are governed by choices related to outcomes beyond specific food-

related outcomes, such as hunger, and include social and emotional outcomes, such as social acceptance and self-esteem.

Sbrocco, Hodges, Gallant and Lewis (1998) found that obese women were not aware of the positive consequences of their eating behaviors or how these positive consequences acted to maintain their problematic eating behavior. Other studies demonstrated that restrained eaters and obese women, unlike normal weight women, typically restricted eating or overate and were generally unable to eat in moderation (Sbrocco & Schlundt, 1998). These data suggest that obese women would benefit from both understanding how they make decisions regarding eating choices and learning to eat in moderation (Sbrocco, Hodges, Gallant, & Lewis, 1998). Using the decision-making model in teaching individuals to understand choices in eating behavior and to promote moderation in eating behavior, BCT is intended to increase long-term weight loss maintenance, as it is associated with promoting more realistic food choices that can be maintained over time.

Traditional behavior therapies encourage individuals to closely follow a moderate caloric prescription of 1000 - 1200 kcal/day and to compensate when they overeat by skipping meals or eating less at meals in order to maintain weight loss. Traditional dieting treatments typically reinforce unrealistic behaviors such as over-restriction and elimination of foods from one's diet, which ultimately maintain the "dieting mentality." As the research suggests, dieting treatments are successful in achieving short-term weight loss; however, they lack long-term effectiveness because of both a biological necessity to increase caloric intake as well as the increased likelihood of abandoning the diet once, and inevitably when, dieting rules are broken.

BCT is different from traditional behavior therapies in several respects. Individuals in BCT are not encouraged to overly restrict their food intake. Instead, individuals are taught to eat realistically by eating foods in a manner consistent with a slower rate of weight loss, but with long-term weight loss maintenance. Individuals in BCT are prescribed a more mild caloric restriction of 1800-2000 kcal/day. Individuals are not only discouraged from compensatory behaviors such as banking calories or meal skipping after periods of overeating, they are taught how to eat reasonable amounts of forbidden and higher fat foods. Participants in BCT are taught how to eat problem foods while also placing emphasis on reasonable weight loss goals. Behavioral Choice Treatment places less restriction on individuals and teaches them to make slow changes that will last over time.

Different rules regarding eating underlie each treatment. Behavior therapy participants learn rigid eating rules that promote the "dieting mentality" as suggested in earlier theories of eating and self-regulation (Brownell & Rodin, 1994; Herman & Polivy, 1980). In adhering to these more rigid rules, it is expected that individuals will restrict both in the amount of food intake as well as the types of foods they eat. Once these rules are broken, dysregulation will occur, leaving the individual vulnerable to break the diet and overeat. BCT, on the other hand, promotes more flexible eating, allowing participants to consume a greater caloric intake as well as a greater variety of foods. In BCT, individuals are taught to make decisions about the cost of eating rather than simply adhering to eating rules; therefore, no eating rules can be broken, in spite of foods consumed. Because there are no strict dietary rules in BCT, the on-diet/off-diet

phenomena is a mute point. Instead, healthy lifestyle changes are considered a life-long process.

In a preliminary study, Sbrocco and colleagues (1999) compared weight change in BCT with a traditional behavioral weight management program (BT). Results demonstrated that weight loss in BCT, although at a slower rate compared to BT, was maintained over time (See Figure 1). The differences found in weight loss between BT and BCT were expected based on their respective focus on promoting rules versus flexibility. We have yet to fully understand the mechanisms behind these differences, however. Investigating eating patterns is one way to examine mechanisms that contribute to weight loss and long-term weight loss maintenance.

Existing Research on Eating Patterns

Few studies to date have examined changes in eating patterns as a result of weight loss treatments. Of those that have, most have used self-report data or retrospective recall of eating behavior rather than prospective self-monitoring, and none have compared eating patterns across obesity treatments. Findings from studies that have examined eating patterns in obese participants, although not directly related to weight change, are important to note. Russ, Ciavarella, and Atkinson (1984) identified several eating behaviors characteristic of obese weight loss participants, including skipping breakfast, frequent snacking, consumption of the largest portion of food after 5 p.m., and lack of stability in eating patterns (i.e., skipping meals some days and overeating other days). Basdevant, Craplet, and Guy (1993) examined snacking patterns in obese women seeking weight loss treatment. Dietary histories indicated that energy intake was not related to body weight, per se, but was related to recent body weight history. Snacking was

characteristic of the sample of obese women, and individuals identified as 'snackers' reported a greater daily energy intake and a greater intake during meals compared to 'non-snackers.' Bjorvell, Ronnenberg, and Rossner (1985) found that treatment seeking overweight women demonstrated large variability in their amount and type of food intake.

Looking at energy intake, Anderson and Rossner (1989) interviewed obese females before and after a weight reduction study. After participating in a weight loss treatment program, two cohorts of participants retrospectively reported a reduction in average daily kilojoules and fat intake and an increase in carbohydrate intake relative to their pre-treatment levels. More recently, Kern et al. (2002) examined self-reported eating patterns among participants in a residential treatment center for weight control and lifestyle change. Analyses of changes in eating patterns revealed a decrease in self-reported snacking, portion size, binging, and skipping meals over time. There were no differences in the amount of change in these factors, however, between participants who lost weight over time and those who did not.

Together, these studies suggest that eating patterns associated with obesity include meal skipping, snacking, and lack of stability in eating patterns. However, whether these eating behaviors generalize to overweight or obese populations, particularly during weight loss, is not known. In the studies noted above, most lacked normal weight or treatment control groups and many lacked baseline data. When eating patterns were examined after a weight loss program, in some cases, there was a lack of standardized treatment among participants. Additionally, methods for assessing eating

patterns involved retrospective history and self-report, including Likert scales to rate the frequency of behaviors, rather than prospective self-monitoring.

Fundamental Issues in Assessment Methods of Eating Patterns

Self-monitoring, a key strategy in behavioral weight loss treatments, is used to aid in both the assessment and treatment phases (Latner & Wilson, 2002). Self-monitoring provides information on eating behaviors, including the types and amounts of foods eaten, the macronutrients associated with consumed foods (e.g., calories and fat), and information regarding the situational and emotional variables associated with food consumption. Assessing eating behaviors can also provide information about how treatment may be impacting changes in eating behaviors.

The issue of methodology for assessing eating patterns is an important one to consider. As noted previously, assessment of eating patterns in previous studies has included self-monitoring food records, retrospective food recall, and food frequency questionnaires. Each method provides slightly different information and has its own limitations in terms of the type of information assessed, the quality of the data, and analytic strategies to obtain nutrient estimates (Thompson & Subar, 2001). Understanding the differences in assessment methods provides the necessary backdrop for truly understanding and interpreting results of studies that have examined eating patterns.

Self-monitoring food records measure dietary intake prospectively, over a specified time period. The strengths of this method include the provision of details about specific foods and amounts that are consumed in the moment and reduced error associated with recall (Thompson & Subar, 2001). This method, however, tends to be

tedious. Self-monitoring requires time to learn how to use self-monitoring records, whether paper and pencil or computerized diaries, and a high level of motivation from users. Research has consistently shown that behavior is highly reactive to the initiation of self-monitoring, such that individuals change the type and amount of food eaten in order to make recording easier or to represent their eating habits in a more positive way. In addition, individuals may make errors in food weighing or in portion size reporting. When energy intake assessed with self-monitoring records is compared with more physiological assessment methods, such as the doubly labeled water technique, energy estimates from self-monitoring tend to be lower than what is required for energy balance (Lissner et al., 1989). This suggests that individuals are either eating less in response to the self-monitoring or they are under-reporting their food intake.

Retrospective food recall, or retrospective dietary history, is a method in which a trained interviewer "asks and probes" individuals to report dietary intake, including the types and amounts of foods and drinks consumed, over 24-hours (24-hour recall) or over the last week or month (history recall). This method is widely used to assess group mean intakes in population-based surveys; however, by its nature, it limits the representation of general dietary intake based on either an assessment over a 24-hour period or a generalization of intake based on recall over several weeks. Here, as seen with self-monitoring, under-reporting is evident (Lichtman et al., 1992) and may be the result of measurement error, response bias, or an error in memory.

Food frequency questionnaires (FFQ) are self-reports of eating behavior (e.g., long lists of food for individuals to endorse whether or not they eat each food, level of hunger before the eating episode, and/or situational variables during which the eating

episode takes place), which often require reporting behavior over a long period of time, even up to a year (Hernandez et al., 1998). The strength of these methods is their practicality, as they are less costly than using self-monitoring or food recall. The limitations of these methods include their lack of specificity in assessing eating behavior (e.g., portion sizes are not assessed) and their vulnerability to recall error.

Self-monitoring food records, retrospective recall, and FFQ's each have their own unique strengths and limitations. In the scientific community, there is an acknowledgement of these limitations, along with a general acceptance of them, as more accurate measures of energy intake such as the doubly labeled water technique, are both costly and time consuming. When using these techniques to examine eating behavior, understanding the benefits and limitations of the assessment measure can add to the validity of interpreting the results.

Purpose of the Current Study

To date, variations of similar weight loss strategies have been examined in weight loss treatment programs. From that research, we know that traditional behavior therapies are successful in promoting notable short-term weight loss, but that weight is regained over time. What we do not know is why these losses cannot be maintained over time. We do not know what mechanisms contribute to the short-term success in traditional treatments or what mechanisms are involved in long-term weight loss maintenance. Few studies to date have examined changes in eating patterns as a result of weight loss treatments. Of those that have, none have used prospective self-monitoring techniques to examine eating patterns across treatment. Perhaps understanding how eating behaviors change across BCT, one of the few treatments known to promote greater weight loss

maintenance compared to a more traditional behavior therapy, will contribute to our pursuit of successful long-term weight loss treatments.

The purpose of the present study was to compare changes in eating patterns across two types of weight management programs: Traditional Behavior Therapy (BT) and Behavioral Choice Treatment (BCT). Because eating behavior will differ depending on rules regarding eating, and because BT and BCT employ different rules for eating, we expect participants' behavior to differ between these treatment groups. Four eating-related variables were investigated: kilojoule[1] intake, fat intake, frequency of eating, and the variability of eating. By analyzing data on eating patterns across dieting treatments, we hope to understand decisions pertinent to eating and the cognitions associated with eating decisions. By identifying how eating patterns change over treatment, we hope to learn how eating behaviors contribute to weight loss and weight loss maintenance so specific patterns that are conducive to managing weight over a lifetime can be delineated. Broadly, it is hypothesized that there will be significant differences in the changes in eating patterns across weight loss treatments and that those changes that occur within the Behavioral Choice Treatment will contribute more significantly to the success of short-term weight loss. More specifically, the parameters indicated above will be analyzed to examine the hypotheses discussed below.

Hypotheses

Specific Aim 1: To determine whether the BT and BCT groups are different at baseline on all variables, including mean daily kilojoule intake, mean percent of calories from

[1] Kilojoules (kJ) measure energy converted from foods such as carbohydrates, proteins, and fats. Kilojoules are commonly called calories or kilocalories, however, 'kilojoule' is a term more internationally accepted when addressing research data. Mathematically, one calorie has the energy value equivalent to 4.186 kJ.

fat (percent fat), mean daily fat grams, and frequency of eating episodes.

Hypothesis 1: Differences at baseline. It was expected that the BT and BCT groups would not differ at baseline in mean daily kilojoule intake, mean daily percent fat intake, or mean daily fat gram intake. Additionally, no differences were expected in mean daily kilojoule intake, mean daily percent fat, or mean daily fat grams for meals, including breakfast, lunch, dinner, and snacks. It was also expected that BT and BCT groups would not differ at baseline in variability of daily kilojoule intake. Lastly, it was expected that BT and BCT groups would not differ in mean daily number of eating episodes or mean number of eating episodes for breakfast, lunch, dinner or snacks.

Specific Aim 2: To determine whether there are differences between BT and BCT in mean daily kilojoule intake across treatment.

Hypothesis 2: Changes in mean daily kilojlue intake at the onset of and as a result of treatment. It was expected that participants in both BT and BCT would reduce mean daily kilojoule intake from baseline (i.e., 2 weeks of self-monitoring prior to treatment) to the beginning of treatment (i.e., the first 3 weeks of treatment). It was further expected that the treatment groups would differ in mean daily kJ intake at the end of treatment (the last 3 weeks self-monitoring data was collected in treatment). More specifically, it was expected that BCT would report a greater mean daily kJ intake compared to BT. Additionally, at the end of treatment, it was expected that BT would maintain a lowered mean daily kJ intake at a level similar to baseline, whereas BCT was expected to report an increase in mean daily kJ intake compared to baseline. These differences were expected for mean daily kJ intake and kJ intake for meals, including breakfast, lunch, dinner, and snacks. Group differences at the middle of treatment (weeks

4-7 of treatment) were examined to identify points of change within treatments, but no a priori hypotheses were made.

These expectations were based on clinical observations that participants, regardless of treatment condition, initiate "dieting" at the onset of treatment as evidenced by a marked reduction in total daily kJ intake. The initiation of dieting at the onset of treatment was determined by the difference in mean daily kJ intake from baseline to the beginning of treatment. The prediction for a group difference at the end of treatment was based on expected adherence to treatment recommendations for continued dieting throughout BT and for learned flexibility surrounding eating decisions in BCT.

Specific Aim 3: To determine whether there are differences in mean daily percent of calories from fat (percent fat) and mean daily fat gram intake between the BT and BCT groups as a result of treatment.

Hypothesis 3: Changes in mean daily percent fat and mean daily fat gram intake at the onset of treatment and as a result of treatment. It was expected that mean daily percent calories from fat (percent fat) and mean daily fat gram intake would decrease from baseline to the beginning of treatment for both BT and BCT. Furthermore, it was expected that the BT and BCT groups would differ in mean percent fat and fat gram intake at the end of treatment. More specifically, it was expected that BCT would report a greater mean daily percent fat and fat gram intake compared to BT. Additionally, at the end of treatment, the BT group was expected to maintain a reduced mean daily percent fat and fat gram intake at levels similar to baseline whereas the BCT group was expected to report a greater mean daily fat intake compared to their baseline levels. These group differences were expected for mean daily percent fat and fat gram

intake and mean daily percent fat and fat gram intake for meals, including breakfast, lunch, dinner, and snacks. Differences at the middle of treatment (weeks 4-7 of treatment) were examined to identify points of change within treatments.

These expectations were based on clinical observations that participants, regardless of treatment condition, initiate restriction of food intake at the onset of treatment by a marked reduction in higher fat and 'forbidden foods.' The initiation of food restriction at the onset of treatment was determined by differences in mean daily percent fat and mean daily fat gram intake from baseline to the beginning of treatment within both BT and BCT. In addition, these expected differences were based on expectations for participant adherence to their respective treatment protocols. Participants in BT were reinforced for restriction whereas participants in BCT were encouraged to be more flexible in their eating behaviors, leading to the proposed group differences in fat intake.

Specific Aim 4: To determine whether BT and BCT differ in variability of daily kJ intake across treatment.

Hypothesis 4: Treatment group differences in variability of daily kJ intake at the beginning of treatment and at the end of treatment. It was expected that the variability of daily kilojoule intake would differ between groups at the end of treatment. More specifically, it was expected that BT would indicate greater variability of daily kJ intake at the end of treatment compared to BCT. These expectations are based on evidence that food avoidance is associated with abstinence rule violation, which often precipitates cycles of restriction followed by periods of over eating (Herman & Polivy, 1980) and observations that participants in both BT and BCT initiate caloric restriction at

the onset of treatment. It is, therefore, expected that both groups will limit their food intake, resulting in kJ intakes that vary little at the beginning of treatment. Additionally, there is evidence that moderate caloric restriction is unrealistic and is found to be difficult to maintain over time (Herman & Polivy, 1980). Because of the assumption in this study that BT, but not BCT, promotes food restriction and avoidance, BT was expected to report greater variability in kJ intake compared to BCT at the end of treatment.

Specific Aim 5: To determine whether there are differences in mean frequency of meals between the BT and BCT groups as a result of treatment.

Hypothesis 5: Treatment group differences in mean daily meal frequency at the beginning of treatment and as a result of treatment. It was expected that the mean daily number of eating episodes would decrease from baseline to the beginning of treatment for both BT and BCT. Furthermore, it was expected that the treatment groups would differ in mean daily number of eating episodes at the end of treatment. More specifically, it was expected that BCT would report a greater mean daily number of eating episodes compared to BT. Additionally, at the end of treatment, it was expected that BT would maintain a decreased mean daily frequency of eating episodes similar to baseline levels, whereas BCT was expected to report an increase in mean daily frequency of eating episodes compared to baseline. These differences were expected for mean daily number of eating episodes and mean daily eating episodes at specific meal times, particularly breakfast and snacks. Differences at the middle of treatment (weeks 4-7 of treatment) were examined to identify points of change within treatments with no a priori hypotheses.

These expectations were based on clinical observations that participants, regardless of treatment condition, initiate “dieting” at the onset of treatment by reducing

their food intake. These efforts towards reduced food intake are believed to increase the incidence of meal skipping, resulting in a decreased number of daily eating episodes. Further hypotheses are based on expectations of treatment adherence. Participants in BCT are encouraged to eat frequently to avoid hunger whereas participants in BT are reinforced for restriction of food intake, skipping meals, and banking calories. These differences were expected for average daily number of eating episodes and specifically expected for number of eating episodes for breakfast and snacks.

Methods

Participants

Study participants were 32 nonsmoking, otherwise healthy obese women who participated in a 13-week weight management program in the Washington, D.C. metropolitan area. Twenty-four participants were part of the original, preliminary study, results of which are described elsewhere (Sbrocco et al., 1999). Participants were required to be 30-60% above ideal body weight as defined by the Metropolitan Life Insurance Company weight charts (1983) and could not have lost more than 10 pounds in the last month or more than 20 pounds in the last 3 months prior to entering the study. At pre-treatment, participants were, on average, 42.4 years of age (SD=8.76), had 15.2 years of education (SD=2.42), and a BMI of 35.7kg/m^2 (SD=5.35). Sixty-eight percent of participants were African American, 28% were Caucasian, and 3.1% were Hispanic.

Procedures

Participants were recruited through advertisements for a weight management study. At an initial orientation session, participants received thorough instruction on use of a computerized self-monitoring eating diary. Computerized self-monitoring diaries,

using Psion 3.0 palmtop computers (Psion PLC, London, England), recorded eating episodes. Participants weighed all foods in grams or ounces using a portable scale. Dietary intake was recorded using the Comcard COMPUTE-A-DIET Nutrient Balance System software program (Comcard, 1993), which contains nearly 4,000 foods from the United States Department of Agriculture (USDA) data base. Participants were required to prospectively enter all eating episodes for a 2-week baseline period, during which they were instructed not to alter their eating patterns or try to lose weight. After completing the baseline period, participants were randomly assigned to either a traditional Behavior Therapy (BT; *n*=16) or Behavioral Choice Therapy (BCT; *n*=16).

All participants were provided with 2-weeks of meal plans, which differed only in kilojoules prescription; BT participants were prescribed a 5,023 kJ/day (1200kcal) intake whereas BCT participants were prescribed an intake of 7534 kJ/day (1800 kcal). Both plans followed a macronutrient composition of 50%-60% carbohydrates, 20%-25% fat, and 15%-20% protein. Both treatment protocols included 13 90-minute sessions co-led by a clinical psychologist and a graduate student in psychology. All participants were weighed weekly on a balance beam scale.

For the first 10 weeks of treatment, computerized self-monitoring food data was collected and downloaded by research assistants. Daily kilocalories, fat, protein, and carbohydrates were evaluated and summarized for participants using individualized graphs and individualized participant feedback on a weekly basis. The use of self-monitoring dairies was terminated after Week 10 of treatment in order to allow participants to adjust to eating and to treatment gains without the diaries while still within the treatment environment.

The 13-week BT protocol was based on a modified version of Brownell's 16-week LEARN program (Brownell, 1997), which is a widely used behavioral weight loss treatment that utilizes techniques such as food avoidance and banking calories. In order to match treatment length and therapist contact with the BCT group, the original 16-week LEARN was condensed into 13-weeks, as depicted in Table1. Participants were reinforced for avoidance of high calorie, high fat foods and for staying within a kilojoule prescription of 5023 kJ/day (1200 kcal/day).

The BCT protocol (Sbrocco et al., 1999) is a skills training program and was designed to achieve slower weight loss and to improve weight loss maintenance through teaching participants strategies, including practicing eating "forbidden foods" and applying moderation and consistency to macronutrient intake. BCT employed a mild kJ restriction of 7500-8300 kJ/day (1800-2000 kcal/day). Participants learned to eat all types of foods in reasonable amounts and to avoid meal skipping or 'saving calories.'

Analytic Strategy

Following standard analytic strategy in the eating and weight literature, group differences in baseline levels and group differences from baseline (2 weeks prior to treatment) to time 1 (beginning of treatment) differences were examined separately from differences between groups across treatment (beginning of treatment: weeks 1-3 of treatment, middle of treatment: weeks 4-7 of treatment, and end of treatment: weeks 8-10 of treatment). The rationale for analyzing baseline to treatment onset separately from the beginning to end of the treatment phase is to capture the hypothesized initiation of dieting with treatment onset, regardless of treatment modality. Including all time points from baseline to the end of treatment in a single repeated-measures analyses would mask the

changes that occur across the treatment phases due to the large expected differences between baseline and the beginning of treatment. This would take away from true purpose of this study, which is to examine the effects after the onset of treatment itself.

For hypothesis 1, ANOVA's were used to examine baseline differences between the treatment groups for mean daily kilojlule intake, fat intake, variability of kilojoule intake, and frequency of eating episodes. For hypothesis 2, 2 (group: BT, BCT) x 3 [time: beginning of treatment (weeks 1-3 of treatment), middle of treatment (weeks 4-7 of treatment), end of treatment (weeks 8-10 of treatment)] repeated-measures ANCOVA, covarying for baseline intake, was used to examine changes in mean daily kilojoule intake and kilojoule intake at meals (breakfast, lunch, dinner, snacks) across treatment. A significant group x time interaction was expected, and would be followed up with a 2-way (group: BT, BCT) ANOVA within baseline, the beginning of treatment, and the end of treatment to examine group differences at these time points. Additionally a 3-way (time: beginning of treatment, middle of treatment, end of treatment) ANCOVA within each group was used to examine changes across treatment for each group, covarying for baseline caloric intake. A main effect of group at the end of treatment was expected, such that BCT would demonstrate greater mean daily kilojoule intake compared to BT. A main effect of time within both groups was expected. It was expected that BCT would demonstrate a greater mean daily kilojule intake from baseline to the beginning of treatment, no differences from the beginning to the middle of treatment, and greater kilojoule intake at the end of treatment compared to the beginning of treatment. It was expected that BT would demonstrate a greater kJ intake from baseline to the beginning of

treatment, no differences from the beginning to the middle of treatment or from the beginning of treatment to the end of treatment.

Similar to the analyses used for hypothesis 2, for hypothesis 3, a 2 (group: BT, BCT) x 3 [time: beginning of treatment (weeks 1-3 of treatment), middle of treatment (weeks 4-7 of treatment), end of treatment (weeks 8-10 of treatment)] repeated-measures ANCOVA, covarying for baseline intake, was used to examine changes in mean daily percent calories from fat and fat gram intake overall and for each meal (breakfast, lunch, dinner, snacks) across treatment. A significant group x time interaction was expected, and would be followed up with a 2-way (group: BT, BCT) x ANOVA within baseline, beginning of treatment and end of treatment to examine group differences at these time points. Additionally, a 3-way (time: beginning of treatment, middle of treatment, end of treatment) ANCOVA within each group to examine changes across treatment for each group, covarying for baseline mean daily percent calorie fat from and fat gram intake. A main effect of group at the end of treatment was expected, such that BCT would demonstrate greater mean daily percent fat and fat gram intake compared to BT. A main effect of time within both groups was expected. It was expected that BCT would demonstrate a greater mean daily percent fat and fat gram intake from baseline to the beginning of treatment, no differences from the beginning to the middle of treatment, and greater percent fat and fat gram intake at the end of treatment compared to the beginning of treatment. It was expected that BT would demonstrate a greater percent fat and fat gram intake from baseline to the beginning of treatment and no differences from the beginning to the middle of treatment or from the beginning of treatment to the end of treatment.

For hypothesis 4, the standard deviation of daily kilojoules, as a measure of variability in food intake, was analyzed for each participant in two time periods: baseline and the end of treatment. Because the data was not normally distributed and rules of independence were violated, a non-parametric test was used. A Mann-Whitney U test was used to examine treatment group differences in variability in daily kilojoule intake (i.e. median standard deviation of kJ intake) at each time point.

Similar to hypotheses 2 and 3, for hypothesis 5, a 2 (group: BT, BCT) x 3 (time: beginning of treatment, middle of treatment, end of treatment) repeated-measures ANCOVA, covarying for baseline intake, was used to examine changes in meal frequency overall and for each meal (breakfast, lunch, dinner, snacks) between groups and across treatment. A significant group x time interaction was expected, which would be followed up with a 2-way (group: BT, BCT) ANOVA at baseline, beginning of treatment, and end of treatment to identify group differences at these points. Additionally, a 3-way (time: beginning of treatment, middle of treatment, end of treatment) ANCOVA, covarying for baseline, in order to examine the main effects of treatment within each group. A main effect of group at the end of treatment was expected, such that BCT would demonstrate greater frequency of meals compared to BT. A main effect of time within both groups was expected. It was expected that BCT would demonstrate a greater frequency of meals at baseline compared to the beginning of treatment, no differences from the beginning to the middle of treatment, and a greater frequency of meals at the end of treatment compared to the beginning of treatment. It was expected that BT would demonstrate a greater frequency of meals at baseline compared to the beginning of treatment, no differences from the beginning to the middle

of treatment and no differences between the beginning of treatment and the end of treatment.

Results

Demographic Information

Demographic information for participants is presented in Table 2. At pretreatment, no significant differences between groups were found for age, $F(1, 30) = 0.056$, $p = 0.82$, weight $F(1, 30) = 0.235$, $p = 0.63$, BMI, $F(1, 30) = 0.191$, $p = 0.67$, level of education, $\chi^2(2, n=32) = .234$, $p = 0.89$, ethnicity, $\chi^2(2, n=32) = 3.03$, $p = 0.22$, or marital status, $\chi^2(3, n=32) = 1.60$, $p = 0.67$.

Specific Aim 1: Baseline Data

Hypothesis 1: Baseline intake.

Table 3 presents mean daily kilojoule intake, and kilojoule intake for each meal, including breakfast, lunch, dinner, and snack. No significant treatment group differences were found between BT and BCT at baseline for mean daily kilojoule intake, $F(1,29) = 1.66$, $p = 0.21$, or for mean daily kilojoule intake at breakfast, $F(1, 29) = .46$, $p = 0.83$, lunch, $F(1,29) = 2.09$, $p = 0.16$, dinner, $F(1,29) = 1.67$, $p = 0.21$, or snack, $F(1,29) = 0.05$, $p = 0.83$.

Table 4 presents mean fat gram intake and Table 5 presents mean daily percent fat intake at baseline, including daily total intake and intake for each meal. No significant differences were found between BT and BCT at baseline for daily fat grams, $F(1,29) = 1.44$, $p = 0.24$, or percent fat intake, $F(1,30) = 3.11$, $p = 0.09$. No significant differences were found between BT and BCT for fat gram intake for breakfast, $F(1, 29) = .376$, $p = 0.55$, lunch, $F(1,29) = 2.27$, $p = 0.12$, dinner, $F(1,29) = .920$, $p = 0.35$, or snack, $F(1,29)$

= .016, p= 0.90. No significant differences were found for percent fat intake for breakfast, $F(1, 30) = .446$, p= 0.51, lunch, $F(1,30) = .097$, p= 0.76, or dinner, $F(1,30) = 1.33$, p= 0.26. At snacks, however, the BCT group reported a greater percent fat intake compared to BT, $F(1,29) = 10.27$, p< .01.

Figure 2 presents the median standard deviation of kilojoule intake at baseline for BT and BCT. The Mann-Whitney U test of two independent comparing the groups on standard deviations of kilojoule intake at baseline revealed no significant differences at baseline between BT and BCT (median = 822.11, p= 0.91). Figure 3 presents the mean daily number of eating episodes and the daily average number of breakfasts, lunches, dinners, and snacks during baseline. No significant differences between BT and BCT were found for number of eating episodes, $F(1,30) =0.34$, p= 0.85, or for number of breakfasts, $F(1, 30) = 1.19$, p= 0.28, lunches, $F(1,30) = 0.07$, p= 0.79, dinners, $F(1,30) = .809$, p= 0.38, or snacks, $F(1,30) = .002$, p= 0.96. Figure 4 presents the mean daily percentage of total meals consumed at baseline for breakfast, lunch, dinner, and snack. No significant differences between BT and BCT were found at baseline for percentage of total meals eaten at breakfast, $F(1, 30) = .667$, p= 0.42, lunch, $F(1,30) = 0.932$, p= 0.34, dinner, $F(1,30) = .1.55$, p= 0.22, or snack, $F(1,30) = .907$, p= 0.35.

Specific Aim 2: Mean Kilojoule Intake

Hypothesis 2: Mean kilojoule intake.

Daily Kilojoule Intake: Mean daily kJ intake by treatment phase (beginning, middle, and end of treatment) for each group is depicted in Figure 5. There was a significant group x time interaction, $F(2, 56)=3.9$, p= 0.03; $\acute{\eta}^2=.0123$, power = .684. As expected, both treatment groups initiated restriction at the onset of treatment such that

they did not differ on kJ intake at the beginning of treatment, despite different kilojoule prescriptions, F (*2,28*) =1.28, p= 0.27. There was a significant decrease in kilojoule intake from baseline to the beginning of treatment within both BT, F(*1, 15*) =18.7, p< .01, and BCT, F(*1, 14*) =35.6, p< .01. After the onset of treatment there was a significant increase in kilojoule intake from the beginning of treatment to the end of treatment for BCT, F(*1, 14*) =5.89, p= 0.03, but not for BT, F(*1, 15*) =1.39, p= 0.26. In spite of this increase for BCT, there was still a significant decrease in kilojoule intake from baseline to the end of treatment, F(*1, 14*) =14.44, p< 0.01.

No significant differences between the treatment groups were found at the middle of treatment, F(*2, 27*) =1.5, p= 0.23. However, at the end of treatment the BCT group reported greater intake compared to BT, F (*2,28*) =5.36, p= 0.03.

Kilojoule intake by meal. Group changes in mean daily kJ intake for breakfast, lunch, dinner, and snack across treatment phase (baseline, beginning, middle, and end of treatment) are depicted in Figures 6-9 respectively. There was a significant group by time interaction for breakfast, F (*2, 56*) =5.89, p< 0.1; $\acute{\eta}^2$=.174, power = .858, and dinner, F (*2,56*) =3.79, p= 0.03; $\acute{\eta}^2$=.119, power = .668. Although not statistically significant, there was a trend for groups to differ over time for snack, F (*2,56*) =2.94, p= 0.06; $\acute{\eta}^2$=.095, power = .551. No group by time interaction was found for lunch, F (*5,56*) = 0.955, p=0.39; $\acute{\eta}^2$=.033, power = .208 .

As expected, there was a significant decrease in mean daily kilojoule intake from baseline to the beginning of treatment at breakfast within BT, F(*1,15*) =4.56, p= 0.05, and within the BCT, F(*1, 14*)=17.8, p< 0.01. Both groups also decreased their mean

daily intake at dinner (BT, $F(1,15)=7.46$, $p= 0.02$ and BCT, $F(1,14)=36.9$, $p< 0.01$), and at snack (BT, $F(1,15) =39.1$, $p< .01$ and BCT, $F(1,14)=6.99$, $p= 0.02$).

Mean daily kilojoule intake at specific meals differed over treatment. There was a significant increase in kilojoule intake from the beginning of treatment to the end of treatment within the BCT group for breakfast, $F(1,14) =5.48$, $p= 0.03$, and lunch, $F(1,14) =8.25$, $p= 0.01$. Even with these increases for BCT, there were still significant decreases in kilojoule intake from baseline to the end of treatment for breakfast, $F(1,14) =4.64$, $p= 0.05$, and lunch, $F(1,14) =15.9$, $p= 0.001$. No significant differences from the beginning of treatment to the end of treatment, however, were found in the BCT group at dinner, $F(1,14) =2.33$, $p= 0.15$, or snack, $F(1,14) =3.03$, $p= 0.10$.

Unlike the BCT group, the BT group continued or maintained kilojoule restriction, as indicated by a further decrease in mean daily kilojoule intake from the beginning of treatment to the end of treatment at breakfast, $F(1,15) =4.28$, $p= 0.05$. There were no significant differences within the BT group from the beginning of treatment to the end of treatment for mean daily kilojoule intake at dinner, $F(1,15) =1.64$, $p= 0.22$, or snack, $F(1,15) =.127$, $p= 0.73$.

Looking at differences between groups at the beginning of treatment, there were no statistically significant group differences for mean kilojoule intake at breakfast, $F(2,28) = 2.09$, $p= 0.16$, lunch, $F(2,28) =0.367$, $p= 0.55$, or dinner, $F(2,28) = 3.54$, $p= 0.07$. Unexpectedly, for snacks, the BCT group did report a greater mean daily kilojoule intake, $F(2,28) = 5.66$, $p= 0.02$. At the middle of treatment, no statistically significant differences were found between groups at breakfast, $F(2,28) = 1.61$, $p= 0.22$, dinner, $F(2,28) = .289$, $p= 0.59$ or snack, $F(2,28) = 0.83$, $p= 0.37$. At the end of treatment,

groups differed at breakfast, F (*2,28*) = 4.61, p= 0.04, and snack, F (*2,28*) = 8.73, p <0.01, with the BCT group eating more at each meal. No significant differences were found for kilojoule intake for lunch, F (*2,28*) =2.02, p= 0.17, or dinner, F (*2,28*) = 0.87, p= 0.36.

Specific Aim 3. Mean Fat Intake

Hypothesis 3: fat intake.

Daily percent fat intake. Mean daily percent fat intake by treatment phase (baseline, beginning, middle, and end of treatment) for each group is depicted in Figure 10. There was a significant group x time interaction on mean daily fat intake, F(*2,56*)=3.68, p= 0.03; $\acute{\eta}^2$=.116, power = .655. As predicted, there was a significant decrease in mean daily percent fat intake from baseline to the beginning of treatment for both BT, F(*1,15*) =21.09, p< .01, and BCT, F(*1,15*) =10.73, p< .01. After the onset of treatment, there were no significant differences in mean daily percent fat intake from the beginning of treatment to the end of treatment for BCT, F(*1,14*) =1.12, p= 0.31, or for BT, F(*1,15*) =.128, p= 0.73. Further analyses revealed, despite the lack of significant differences from the beginning of treatment to the end of treatment, that the BT group reported a significant reduction in mean daily percent fat intake from baseline to the end of treatment, F(*1,15*) = 16.33, p< 0.01. This was not true for the BCT group.

Looking at differences in mean daily percent fat intake between groups, in spite of the decrease for both groups from baseline to the beginning of treatment, the BCT group reported a significantly greater intake than BT at the beginning of treatment, F(*2,29*)=5.55, p= 0.03. There were no significant differences between groups at the

middle of treatment, $F(2, 28) = .353$, $p = 0.58$. At the end of treatment, the BCT group reported greater percent fat intake compared to the BT group, $F(2,28) = 11.83$, $p < 0.01$.

Percent fat intake by meal. Group changes in mean daily percent fat intake for each meal across treatment phase (baseline, beginning, middle, and end of treatment) are depicted in Figures 11-14. No significant group by time interactions were found for breakfast, $F(2,56) = 2.01$, $p = 0.14$; $\acute{\eta}^2 = .067$, power = .398, or snack, $F(5,56) = 0.615$, $p = 0.54$; $\acute{\eta}^2 = .021$, power = .148. Although not statistically significant, there was a trend for group x time interaction for lunch, $F(2,56) = 2.69$, $p = 0.07$, $\acute{\eta}^2 = .095$, power = .513. There was a group x time interaction for dinner, $F(2,56) = 4.43$, $p = 0.02$; $\acute{\eta}^2 = .137$, power = .740.

When examining changes at the onset of treatment, there was a significant decrease in mean daily percent fat intake from baseline to the beginning of treatment at breakfast for both BT, $F(1,15) = 12.3$, $p < .01$, and BCT, $F(1,15) = 6.28$, $p = 0.02$, an increase from baseline to the beginning of treatment at lunch for BT, $F(1,15) = 7.27$, $p = 0.02$, but not for BCT, $F(1,15) = 2.38$, $p = 0.14$, a decrease from baseline to the beginning of treatment at dinner for BT, $F(1,15) = 12.73$, $p < .01$, but not for BCT, $F(1,15) = 36.9$, $p = 0.22$, and a decrease from the baseline to the beginning of treatment at snack for both BT, $F(1,15) = 7.95$, $p = .01$, and BCT, $F(1,15) = 12.43$, $p < .01$.

Examining the effects of treatment on mean daily percent fat intake at meals, there were within-group changes from the beginning of treatment to the end of treatment. Within the BCT group, no significant differences were found from the beginning of treatment to the end of treatment at lunch, $F(1,14) = 1.65$, $p = 0.22$, dinner, $F(1,14) = .314$, $p = 0.58$, or snack, $F(1,14) = 2.17$, $p = 0.16$. There were also no significant differences in

the BCT group in mean percent fat intake from baseline to the end of treatment for lunch, $F(1,14) = .063$, $p = 0.81$, dinner, $F(1,14) = .743$, $p = 0.40$. There was, however, a decrease in percent fat from baseline to the end of treatment at snack in BCT, $F(1,14) = 18.86$, $p < 0.01$.

Analyses within the BT group revealed the continuation or maintenance of mean percent fat restriction after the onset of treatment as indicated by no difference in percent fat intake at lunch, $F(1,15) = .164$, $p = 0.69$, dinner, $F(1,15) = .023$, $p = 0.88$, or snack, $F(1,15) = .285$, $p = 0.60$. There was a decrease in percent fat intake at the end of treatment compared to baseline for breakfast, $F(1,15) = 8.99$, $p < 0.01$, lunch, $F(1,15) = 8.69$, $p = 0.01$, dinner, $F(1,15) = 8.94$, $p < 0.01$, and snack, $F(1,15) = 14.2$, $p < 0.01$.

Looking at between-group differences at the beginning of treatment, no significant differences were found for percent fat intake at snack, $F(2,29) = 2.27$, $p = 0.14$. Unexpectedly, the BCT group reported a greater percent fat intake at the beginning of treatment than the BT group at lunch, $F(2,28) = 3.95$, $p = 0.05$, and dinner, $F(2,29) = 4.41$, $p = 0.04$.

For the BCT group at the middle of treatment, no significant between-group differences were found at the middle of treatment for lunch, $F(2,29) = .541$, $p = 0.47$, dinner, $F(2,29) = .397$, $p = 0.53$, or snack, $F(2,29) = 0.179$, $p = 0.68$. At the end of treatment, the groups differed at breakfast, $F(2,28) = 9.79$, $p < 0.01$, lunch, $F(2,28) = 15$, $p < 0.01$, and dinner, $F(2,28) = 8.59$, $p < 0.01$, with the BCT reporting a greater percent fat intake at each meal. No significant differences were found between BT and BCT at the end of treatment for snack, $F(2,28) = 1.19$, $p = 0.28$.

Daily fat gram intake. Mean daily fat gram intake by treatment phase (baseline, beginning, middle, and end of treatment) for each group are depicted in Figure 15. There was a significant groups x time interaction, $F(2,56)=5.59$, $p= .01$; $\acute{\eta}^2=.16$, power = .819.

There was a significant decrease in mean daily fat gram intake from baseline to the beginning of treatment for both BT, $F(1,15) =38.67$, $p< .01$, and BCT, $F(1,14) =39.46$, $p< .01$. After the onset of treatment, there was a significant increase in daily fat gram intake from the beginning of treatment to the end of treatment for BCT, $F(1,14) =6.58$, $p= 0.02$, but no significant changes for BT, $F(1,15) =.061$, $p= 0.81$. Despite both the lack of change in BT and the increase in fat gram intake for BCT over treatment, a significant decrease from baseline to the end of treatment remained for both BT, $F(1,15) = 42.04$, $p< 0.01$, and for BCT, $F(1,14) = 4.36$, $p= 0.05$.

There were no significant differences between groups for mean daily fat gram intake at the beginning of treatment, $F(2,28) =1.02$, $p= 0.32$, or at the middle of treatment, $F(2, 28) =.323$, $p= 0.58$. As expected, at the end of treatment, the BCT group reported greater fat gram intake compared to BT, $F(2,28) =11.25$, $p< 0.01$.

Fat gram intake by meal. Group changes in mean daily fat gram intake for each meal across treatment phase (baseline, beginning, middle, and end of treatment) are shown in Figures 16-19. Groups differed over time for breakfast, $F(2,56) =5.59$, $p< 0.01$; $\acute{\eta}^2=.166$, power = .838, and dinner, $F(2,56) =4.93$, $p= 0.01$; $\acute{\eta}^2=.15$, power = .787. No group significant group by time interaction was found for lunch, $F(2,56) =1.75$, $p= 0.2$, ; $\acute{\eta}^2=.059$, power = .352 or snack, $F(5,56) = 1.11$, $p= 0.34$; $\acute{\eta}^2=.038$, power = .235.

When examining changes at the onset of treatment, there was a significant decrease in fat gram intake from baseline to the beginning of treatment at breakfast for

both BT, $F(1,15) = 20.37$, $p < 0.01$, and BCT, $F(1,14) = 11.93$, $p < 0.01$, and a decrease at dinner for both BT, $F(1,15) = 5.98$, $p = 0.03$, and BCT, $F(1,14) = 16.77$, $p < 0.01$.

Examining the effects of treatment on mean daily fat gram intake, there were within-group differences over treatment. Within the BCT group, there was a significant increase in fat grams from the beginning of treatment to the end of treatment at breakfast, $F(1,14) = 16.19$, $p < 0.01$. For BCT, no significant differences were found over treatment at dinner, $F(1,14) = 3.12$, $p = 0.10$, or at snack, $F(1,14) = .44$, $p = 0.52$. No significant differences were found in BCT for fat gram intake from baseline to the end of treatment at breakfast, $F(1,14) = 3.96$, $p = 0.87$, or at dinner, $F(1,14) = 1.95$, $p = 0.19$.

For BT, no difference in mean daily fat gram intake from the beginning of treatment to the end of treatment were found at breakfast, $F(1,15) = 1.93$, $p = 0.19$, or at dinner, $F(1,15) = .576$, $p = 0.46$. There were decreases in mean fat gram intake at the end of treatment compared to baseline for breakfast, $F(1,15) = 21.03$, $p < 0.01$, and dinner, $F(1,15) = 10.18$, $p < 0.01$.

Looking at between-group differences at the beginning of treatment, there were no significant differences for mean daily fat gram intake at breakfast, $F(2,28) = .027$, $p = 0.87$, or at dinner, $F(2,28) = .013$, $p = 0.91$. At the middle of treatment, the BCT group reported a greater mean daily fat gram intake than the BT group for breakfast, $F(2,28) = 4.44$, $p = 0.04$. No significant differences between groups were found at the middle of treatment for fat gram intake at dinner, $F(2,28) = .929$, $p = 0.34$. At the end of treatment groups differed in fat gram intake at breakfast, $F(2,28) = 13.11$, $p < 0.01$, and at dinner, $F(2,28) = 4.56$, $p = 0.04$, with the BCT group reporting a greater fat gram intake at each meal.

Specific Aim 4. Variability of Kiljoule Intake

Hypothesis 4: variability of food intake.

Figure 20 shows day to day kilojoule variability for each participant in each group. The mean standard deviation ranged from 133.33 – 2252.51 kJ, with a median value of 677 kJ. Changes in standard deviations of kilojoule intake by group over treatment are shown in Figure 21. At the end of treatment, contrary to our expectations, variability is slightly higher for BCT compared to BT; however, this difference is not statistically significant (median = 592.02, $p = 0.07$).

Specific Aim 5: Frequency of Eating Episodes

Hypothesis 5: number of eating episodes.

Daily number of eating episodes. Mean daily number of eating episodes by treatment phase (baseline, beginning, middle, and end of treatment) for each group are depicted in Figure 22. Contrary to expectations, analyses revealed no significant group x time interaction, $F(2,58)=.498$, $p = .61$; $\acute{\eta}^2=.017$, power = .128.

Eating episodes by meal. Group changes in mean daily frequency of eating episodes for each meal across the treatment phase (baseline, beginning, middle, and end of treatment) are depicted in Figures 23-26. There was no group x time interaction at breakfast, $F(2,58) =.800$, $p = 0.45$, at lunch, $F(2,58) =.089$, $p = 0.92$, at dinner, $F(2,58) =.856$, $p = 0.43$, or at snack, $F(5,58) = 0.2.38$, $p = 0.1$.

Discussion

Implications

Despite different caloric recommendations, both Behavior Therapy (BT) and Behavior Choice Therapy (BCT) participants initiated dieting behaviors at the onset of

treatment, as reflected by the significant decrease in both caloric and fat intake from baseline to the beginning of treatment in both treatment conditions. The initiation of dieting in both treatments suggests that there may be a wide-spread and ingrained acceptance of restriction as the standard weight loss method in our culture. That is, moderate and arguably unrealistic levels of restriction may be the default method of weight loss. This may be related to the immediate reinforcement associated with greater restriction in terms of more rapid weight loss. However, it is difficult to maintain moderate caloric restriction for a prolonged period of time (Schlundt, Sbrocco, & Bell, 1989). More mild restriction in BCT was associated with a slower rate of weight loss over treatment, yet there was greater long-term weight loss maintenance out to 2 years of follow up (Sbrocco et al., 1999). The moderate level of caloric restriction recommended in traditional behavior therapies may be responsible for both the greater short-term weight loss and the long-term failure of weight loss maintenance.

Overall, participants in the BT group maintained a lowered caloric and fat intake across treatment whereas participants in the BCT group significantly increased their caloric and fat intake at the end of treatment. These results, along with the differences in weight loss maintenance over time found in BCT compared to BT (Sbrocco et al., 1999) suggest that there are different consequences of mild versus moderate energy deficits. It appears that mild caloric restriction recommendations are more realistic. Therefore, mild rather than moderate caloric restriction may be more manageable over time, leading to greater long-term weight loss and weight loss maintenance over time.

It is not clear at this point whether energy intake alone contributes to long-term weight loss and weight loss maintenance. Cognitive mechanisms may also be a factor in

the ability to adhere to treatment prescriptions and the ability to maintain weight loss over time. In this study, it was assumed that the dieting mentality is reinforced in BT whereas new rules regarding eating are learned in BT. Variability of caloric intake was used in an effort to indirectly differentiate cognitive mechanisms between treatments. Variability in caloric intake may be a measure of the flexibility promoted in BCT, allowing for a broad range of food choices unlike the strict food rules found in BT. There was a trend for the BCT group to exhibit greater variability in caloric intake compared to BT. Further study of these phenomena is needed. Given our findings, flexibility in eating behavior rather than rigid eating rules may be more realistic for long-term success at weight management.

At what point, however, does restriction of energy intake become unrealistic? At what point do rules regarding eating become unrealistic? These questions remain unanswered. Conceptually, the consequences of energy over-restriction have been addressed in the self-regulation literature, including restraint theory (Herman & Polivy, 1980) and the relapse prevention model (Cummings, Gordon, & Marlatt, 1980). The purpose of these models is to understand factors that impact adherence. These factors are then targeted in order to help individuals adhere to the targeted change. Although we have learned a great deal from the work on eating and self-regulation over the last several decades, we have not moved past the dilemma of lack of long-term weight loss success in treatments for obesity. We have not been successful in helping individuals adhere to moderately restricted diets over a longer time. The results of this study suggest a slightly different approach to understanding problems in behavior maintenance. That is, it is important to identify at what point a behavior is too 'costly' or difficult to maintain. The

key questions for weight loss center around the establishment of reasonable energy decrements and reasonable rules for behavior change. For example, at what level of restriction do individuals begin to struggle to maintain behavior change? This question needs to be addressed both physiologically and cognitively in order to understand why differences in BT compared to BCT lead to differences in long-term weight loss maintenance.

Both physiological and cognitive consequences exist as a result of both caloric restriction and rules promoted in either BT or BCT. The moderate caloric restriction in BT may leave participants physiologically hungry. In addition, the rigidity of the dietary rules in BT may leave participants feeling deprived; consequently, individuals are likely to break these rules. Hunger, perception of deprivation, and rule-breaking can all lead to dysregulation and increase the likelihood of overeating and abandonment of "the diet" (Brownell & Rodin, 1994). Alternatively, participants in BCT are less likely to feel hungry because of more mild caloric restriction recommendations. Additionally, unlike the rigid rules in BT, BCT promotes moderation of food intake; therefore, participants learn greater flexibility in food choices. It is much more difficult to break rules that promote moderation. Both mild caloric restriction and flexibility in eating may decrease the likelihood of dysregulation and, therefore, promote eating behaviors that are more manageable over time.

Physiological Energy Decrements: How Little is Too Little?

Individuals in BT decreased their caloric and fat intake from baseline to the onset of treatment and throughout treatment. The BCT participants also decreased caloric and fat intake from baseline to the onset of treatment, but actually increased both over the

treatment phase. Interestingly, there were no differences between the treatment groups in the number of eating episodes or types of meals consumed. The differences between the groups were in the amount and types of food consumed at meals, which suggests that the BT group achieved restraint by restricting overall intake rather than number of eating episodes. This also suggests that meal skipping was not frequently used as a method of compensation in the BT group. Again, these findings for eating patterns, along with the long-term results on weight change in BCT (Sbrocco et al., 1999), suggest that a moderate energy restriction may be unreasonable for people to maintain over a long period of time. These findings are specific to moderate caloric restriction, defined as approximately 1000 – 1200 kcal/day for females, which is the objective of most traditional weight loss programs. Importantly, this level of restriction is similar to the goal for most individuals as they attempt to control their weight.

Some energy deficit is needed in order for weight loss to occur. However, physiologically, certain levels of energy intake have been defined as overly-restrictive in that they may induce a conservation response. The typical woman in this study was 42 years old, weighed 211 lbs and was 5'5" tall, and required approximately 2200 kcal/day to maintain her weight, depending on her activity level (sedentary-light). Therefore, reducing energy intake to 1200 kcal/day decreases food intake by almost 50%. Again, this is the level typically recommended in traditional behavior therapies. The World Health Organization has defined starvation in developing countries as 1200 kcal/day (WHO, 1985). Traditional behavior therapies, therefore, are promoting caloric recommendations at near starvation levels. The dangers of eating below one's energy requirements, first addressed by Nisbett (1972), include hunger, which makes individuals

more vulnerable to eat in response to external cues and to overeat. Therefore, a more mild restriction recommendation, defined as 1800 – 2000 kcal/day for our average participant, is more ideal for long-term weight loss and weight loss maintenance.

Energy recommendations in weight management programs should be individual-based rather than using a set kcal level. Although this may seem obvious, most group treatments or popular "diets" recommend caloric levels that are the same for all women, regardless of weight status. According to the NHLBI Obesity Initiative Task Force (2000), overweight is defined as BMI of 25 to 29.9 kg/m^2 and obesity as is defined as a BMI of 30 kg/m^2 and above. In spite of the different classes of obesity, however, caloric prescription recommendations for groups are circumscribed in the treatment protocol rather than being based on individual needs.

Beyond weight loss and weight loss maintenance, the implications of differences in energy requirements according to weight extend to treatment adherence. Adhering to an energy deficit of 500 kcal/day is very different than a treatment that promotes a deficit of two times that. Given that the typical restriction in traditional behavioral treatments is between 1000 -1200 kcal/day, it is no wonder that the more obese drop out of treatment more often and are less successful in losing weight and maintaining weight loss (NIH, 1998). This phenomenon is generally perceived as indicative of a dose-response relationship between pathology and obesity - the more obese, the more problems. It remains to be seen if more mild recommendations improve adherence among the more obese.

Cognitive Restriction: The Dieting Mentality

Restriction extends beyond actual caloric deficit to include more cognitive consequences related to perception of deprivation and rules promoted in behavioral weight loss treatments. Although treatment groups in this study did not differ significantly in variability of caloric intake, there was a trend for the BCT group to exhibit more variability in caloric intake. This was actually a surprise, as variability of caloric intake was conceptualized as an indicator of dysregulation (typically resulting from the "dieting mentality)." It was hypothesized that variability in eating patterns would be associated with more traditional behavior therapies, and, therefore, associated with lack of weight loss success. Participants in the BT group were expected to report periods of significant food restriction, including restricting overall energy intake and types of foods consumed, due to adherence to the strict rules promoted in BT. Because the rules promoted in BT are considered unrealistic, it was expected that participants would not be able to maintain behaviors associated with such rules; therefore, periods of overeating would follow periods of restriction. This cycle of overeating followed by restriction was thought to be illustrative of behavioral consequences of dysregulation associated with the dieting mentality.

The cycle of over-restriction followed by overeating within the BT group was predicted based on the restraint theory (Herman & Polivy, 1980) and the abstinence violation effect (Cummings, Gordon, & Marlatt, 1980). The restraint theory proposes that once restrained eaters (defined as individuals who are concerned with their weight and who engage in severely restricting their food intake) break their eating rules, or their efforts to restrict intake are interrupted by some disinhibitor, they come to lack any restraint at all. The abstinence rule violation model suggests that individuals who over-

restrict have an increased potential to violate food abstinence rules. Once a situation occurs in which these rules are broken, global negative cognitive and emotional responses follow, and dietary rules are abandoned all together. These predictions were specific to the BT group based on the rigid rules and moderate calorie restriction emphasized in this treatment whereas the BCT group was expected to have greater flexibility in their eating "rules" for both food intake and choices in types of food eaten.

As noted above, the treatment group differences on our measures of variability, although not statistically significant, showed a trend in the direction contrary to expectations. As an index of cycles of periods of over restriction and overeating, this study measured standard deviations of kilojoule. However, we may not have been measuring variability as we intended, or perhaps variability was equal across groups even though the groups engaged in different behaviors. For example, participants in BCT are encouraged to eat all types of food in moderation and discouraged from compensating by meal skipping or under-eating. It may be that variability in BCT is an indication of this flexibility in food intake rather than the dysregulation that results from the abstinence violation effect. Also, variability in the BT group may be the result of cycles of over-restriction followed by overeating due to rigid eating rules.

It is possible that our concept of variability and how it relates to weight loss is inaccurate. Moderate restriction and strict rules promoted in more traditional behavior therapies, similar to BT, lends itself to unrealistic rules. When these rules are broken, periods of restriction are replaced by periods of overeating. Furthermore, more mild caloric restriction and the promotion of flexibility in eating choices results in exercising greater moderation in eating behaviors. These behaviors are more likely to be maintained

over a lifetime. The findings of this study could indicate that variability, in terms of periods of overeating followed by restriction, is more characteristic of BCT compared to BT, bringing into question conceptual differences in treatment. However, given that BCT reported greater caloric and fat intake across treatment, it is more likely that the unexpected group differences in variability were due to a mismatch between how variability was defined and measured and how variability was conceptualized within treatments. Examining such mechanisms will be necessary to further our understanding of the psychological and cognitive mechanisms involved in weight loss treatments and how they lead to eating behaviors that contribute to long-term weight loss success.

Limitations

Several limitations of this study prevent us from fully understanding the mechanisms involved in eating patterns that contribute to long-term weight loss success. First, data in this study were only collected at baseline and over the first 10 weeks of treatment. Data were not collected for the full 13 weeks of treatment or throughout the follow up period. Without this we can only hypothesize about differential changes between treatment groups over time. As we have seen in the results from the first 10 weeks of treatment, changes were found in some variables across treatment, each measured at time points that contained data from 2 weeks (baseline), 3 weeks (the beginning and end of treatment), and 4 weeks (the middle of treatment). This suggests that further changes or additional changes could be present during the last 3 weeks of treatment, which was not measured in this study. Furthermore, because the lack of long-term success represents a fundamental problem in behavioral therapies for weight loss, and because there is evidence that participants in BCT maintain their weight loss over

time, future studies should examine eating patterns over follow up periods to provide us with information to elucidate those eating behaviors that aid in long-term weight loss success.

A second limitation is that certain dimensions of eating behaviors, such as environmental and emotional stimuli for eating and binge eating, were not assessed. The impact of external and emotional stimuli on eating behaviors should not be underestimated. As noted previously, environmental or emotional variables can act as dietary disinhibitors and can lead to overeating (Heatherton, Polivy, & Herman, 1990). More specifically, data on changes in variables such as subjective reports of hunger, overeating, loss of control, and mood, as well as place and company during the eating episode will provide a more complete understanding of obstacles and positive changes related to different obesity treatments. This will aid in a more thorough understanding of the behaviors and mechanisms involved in more long-term weight loss success.

Finally, several factors in this study may limit the generalizability of the current results, including the weight requirements, gender, level of education, and ethnicity of the participants. To participate in this study, individuals were required to be 30%-60% overweight. Although there is currently no evidence to support a difference, findings may not be true for individuals who are less than 30% or more than 60% overweight. All participants in the current study were women. Also, the typical participant in this study had a college education. This leaves us with the question of how different obesity treatments would affect eating patterns for male participants and for participants with lower levels of education. Lastly, although both African American women and

Caucasian women were represented in this study and ethnic groups were equally distributed across the treatment groups, data were not analyzed by ethnicity.

Results may also be limited by the relatively small sample size. The question is whether a larger sample size would reveal additional findings that augment our understanding of how eating patterns may differ between types of obesity treatments. In spite of this, these data provide preliminary evidence for the importance of moderation in food intake, not restriction, when adopting long-term eating behaviors that contribute to weight loss and weight loss maintenance.

Further analyses are necessary in order to gain a more in depth understanding of differences in the variability of eating behaviors across weight loss treatments. Additional studies should examine conditional probabilities (eg., Johnson, Corrigan, Schlundt, & Dubbert, 1990; Schlundt, Sbrocco & Bell, 1989) to determine whether periods of overeating are associated with traditional behavior therapies and how these periods may affect weight loss maintenance. It will also be important to identify whether flexibility in eating behaviors is more characteristic of BCT than BT and how this flexibility may contribute to long-term weight loss.

This study lends support for replacing weight loss programs that promote moderate caloric restriction and dieting restraint (e.g., fewer types of foods consumed) with programs that promote slight caloric restriction and more flexibility in eating. These data suggest that we can teach obese women to adopt new eating patterns, involving small, but lasting changes. Although the BCT group over-restricted in the beginning weeks of treatment, they increased their caloric and fat intake over the remainder of treatment. Our longer-term data on weight change and maintenance (Sbrocco et al, 1999)

suggest that these patterns may be sustainable after treatment completion. Individuals in BCT continued to lose weight out to 2 years. What is still unclear at this point, however, is what differences exist in eating patterns between BT and BCT at the follow up periods. We can assume, based on evidence of continued weight loss, that the acute changes in eating patterns with BCT are likely maintained over time. Studies involving food restriction, without 'dieting' per say, have shown that body weight returns to baseline levels after the cessation of food restriction (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950). The weight regain observed in the BT group, therefore, is likely due to the abandonment of treatment recommendations and a return to baseline eating patterns.

The differences in the maintenance versus abandonment of treatment recommendations may be due to the slight rather than moderate caloric restriction and to the greater flexibility in food choices promoted in BCT compared to BT. The recommendations in the BCT protocol are more realistic and more manageable, conducive to lifestyle changes that can be adopted over a life time. In contrast, the recommendations in BT are less realistic and less manageable, making them difficult to sustain over time. Consequently, when the treatment recommendations for eating are no longer followed, participants return to their baseline eating habits and, ultimately, to their baseline weight.

Conclusions

This study provides preliminary data on the eating patterns associated with different treatments for obesity, laying the ground work for understanding factors that may contribute to long-term weight loss maintenance. Few studies to date have examined the mechanisms involved in successful weight loss and weight loss

maintenance across treatments for obesity. From research over the last several decades, it is clear that dieting treatments, although effective in producing initial weight loss, fail to maintain or promote further weight loss over the long-term (Perri, 1998). BCT has shown improved weight maintenance over traditional behavioral approaches (Sbrocco et al., 1999). By examining eating patterns in BCT compared to BT, these data suggest that slight rather than moderate caloric restriction and eating throughout the day may be more realistic and promote greater success at long-term weight management. Perhaps these observed eating patterns found in BCT allow individuals to better manage eating behaviors in a way that not only contributes to weight loss, but also facilitates weight loss maintenance over time.

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Table 1. Behavior Therapy: Adapting the LEARN Program to 13 sessions

Session 1	Introduction & Lesson 1
Session 2	Lessons 2 & 3
Session 3	Lesson 4
Session 4	Lesson 5
Session 5	Lessons 6 & 7
Session 6	Lesson 8
Session 7	Lesson 9 & 10
Session 8	Lesson 11
Session 9	Lesson 12
Session 10	Lessons 13 & 14
Session 11	Lesson 15
Session 12	Lesson 16
Session 13	Review and Appendix A

Table 2. Demographic Information by Treatment Group

	Behavior Therapy **Mean (SD)**	**Behavior Choice Treatment** **Mean (SD)**
Age (in years)	42.8 (7.23)	42.1 (10.1)
Body Weight (kg)	96.23 (14.24)	96.08 (12.18)
Body Mass Index (BMI: kg/m^2)	36.14 (5.15)	35.31 (5.63)
Ethnicity	African American 69% Caucasian 25% Hispanic 6%	African American 69% Caucasian 31% Hispanic 0
Marital Status	Single 19% Married 69% Divorced 12%	Single 19% Married 50% Divorced 31%
Education	High-School 31% College 44% Advanced 25%	High-School 37% College 44% Advanced 19%

Table 3. Mean Daily Kiolojoule Intake at Baseline by Meal

	Behavior Therapy **Mean (SD)**	**Behavior Choice Treatment** **Mean (SD)**
Daily Intake (kJ)	9,121 (2,550)	10,312 (2,571)
Breakfast (kJ)	1,875 (671)	1,925 (634)
Lunch (kJ)	2,699 (960)	3,199 (967)
Dinner (kJ)	3,168 (1,017)	3,627 (957)
Snack (kJ)	1,751 (554)	1,693 (894)

Table 4. Mean Daily Fat Gram Intake at Baseline by Meal

	Behavior Therapy **Mean (SD)**	**Behavior Choice Treatment** **Mean (SD)**
Daily Intake (gm)	66 (23)	76 (22)
Breakfast (gm)	13 (6)	14 (8)
Lunch (gm)	19 (7)	23 (8)
Dinner (gm)	23 (11)	26 (10)
Snack (gm)	12 (8)	12 (7)

Table 5. Mean Daily Percent Fat Intake at Baseline by Meal

	Behavior Therapy M% (SD)	**Behavior Choice Treatment M% (SD)**
Daily Intake (%)	29% (5%)	32% (4%)
Breakfast (%)	25% (7%)	27% (9%)
Lunch (%)	32% (10%)	32% (6%)
Dinner (%)	31% (8%)	34% (6%)
Snack (%)	26% (6%) [a]	35% (8%) [a]

[a] Denotes statistically significant differences between group at the $p < .01$ level.

Figure 1: Weight Lost (in lbs.) Across Groups Over a 4 year Follow-up

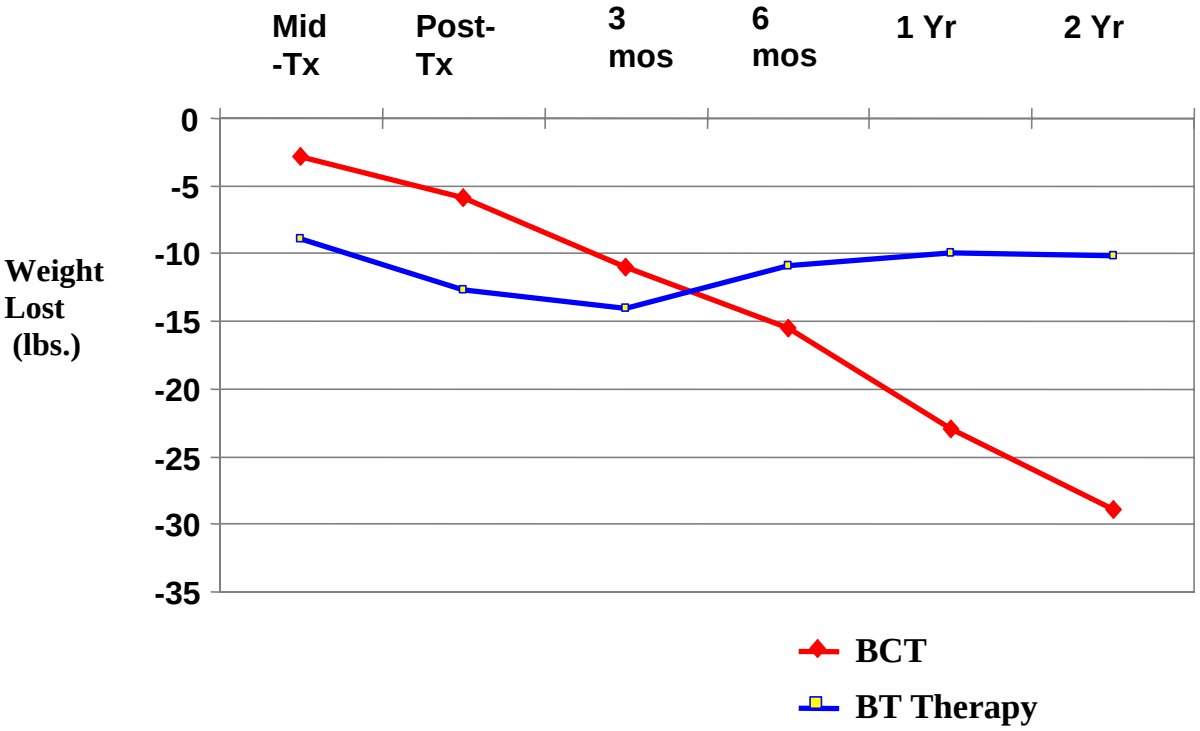


Figure 2. Median Standard Deviation of Daily Kilojoule Intake at Baseline

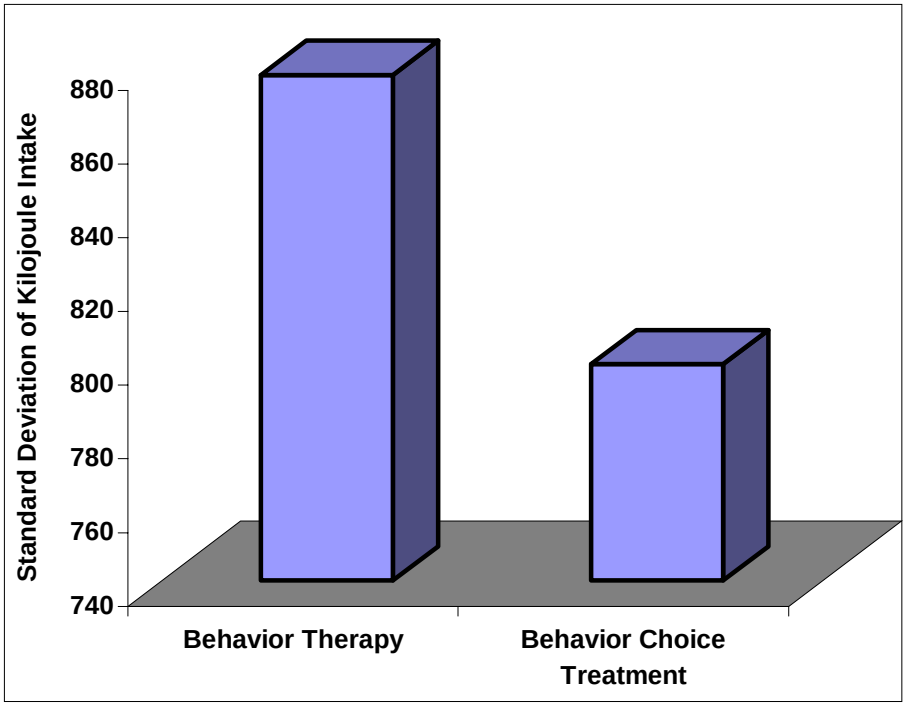


Figure 3. Mean Daily Frequency of Eating Episodes by Group at Baseline

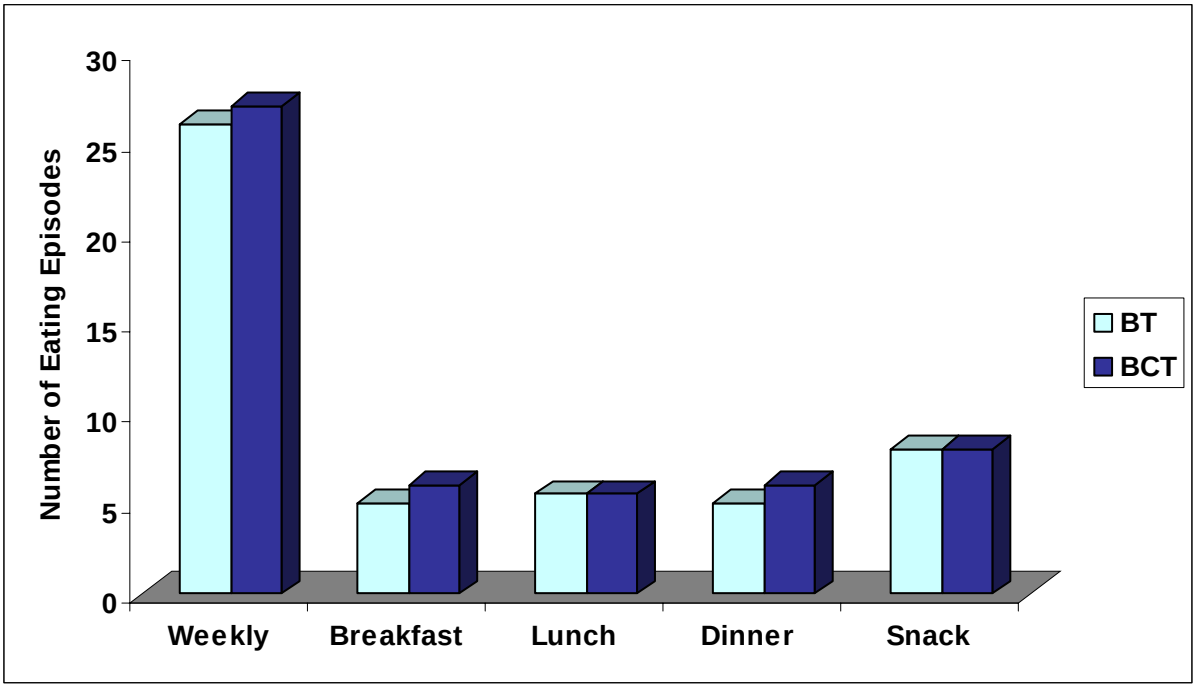


Figure 4. Mean Daily Number of Total Meals by Group During Baseline

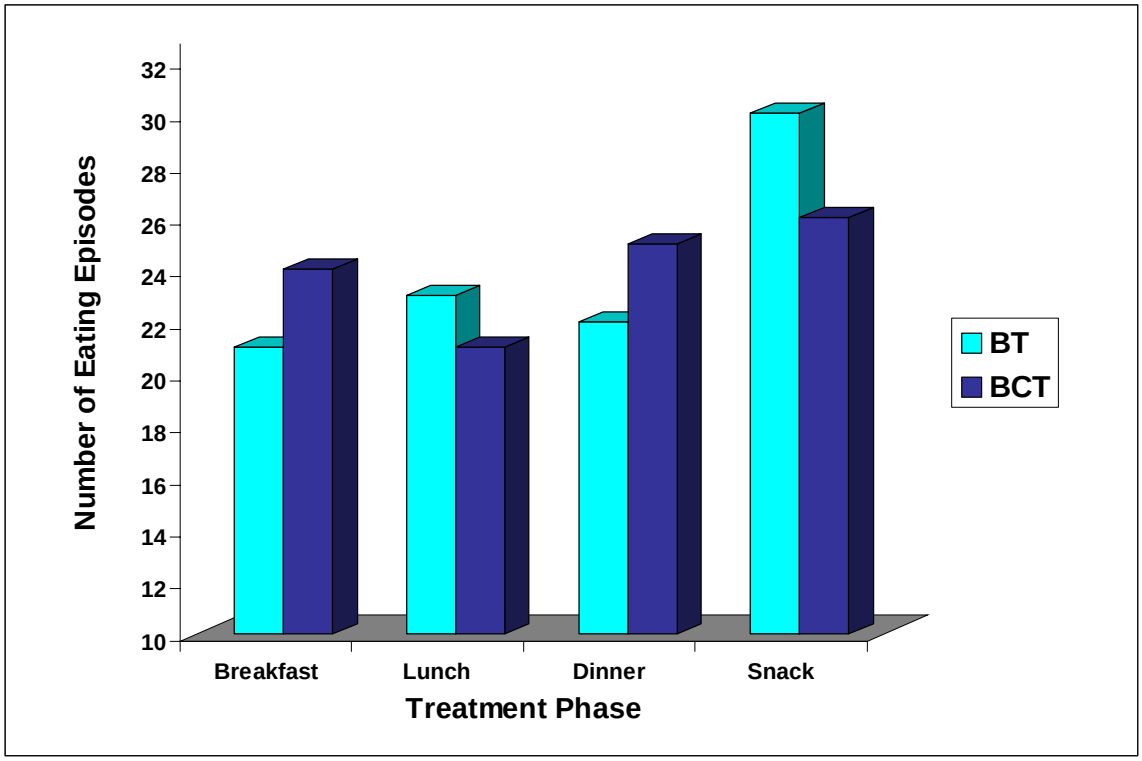
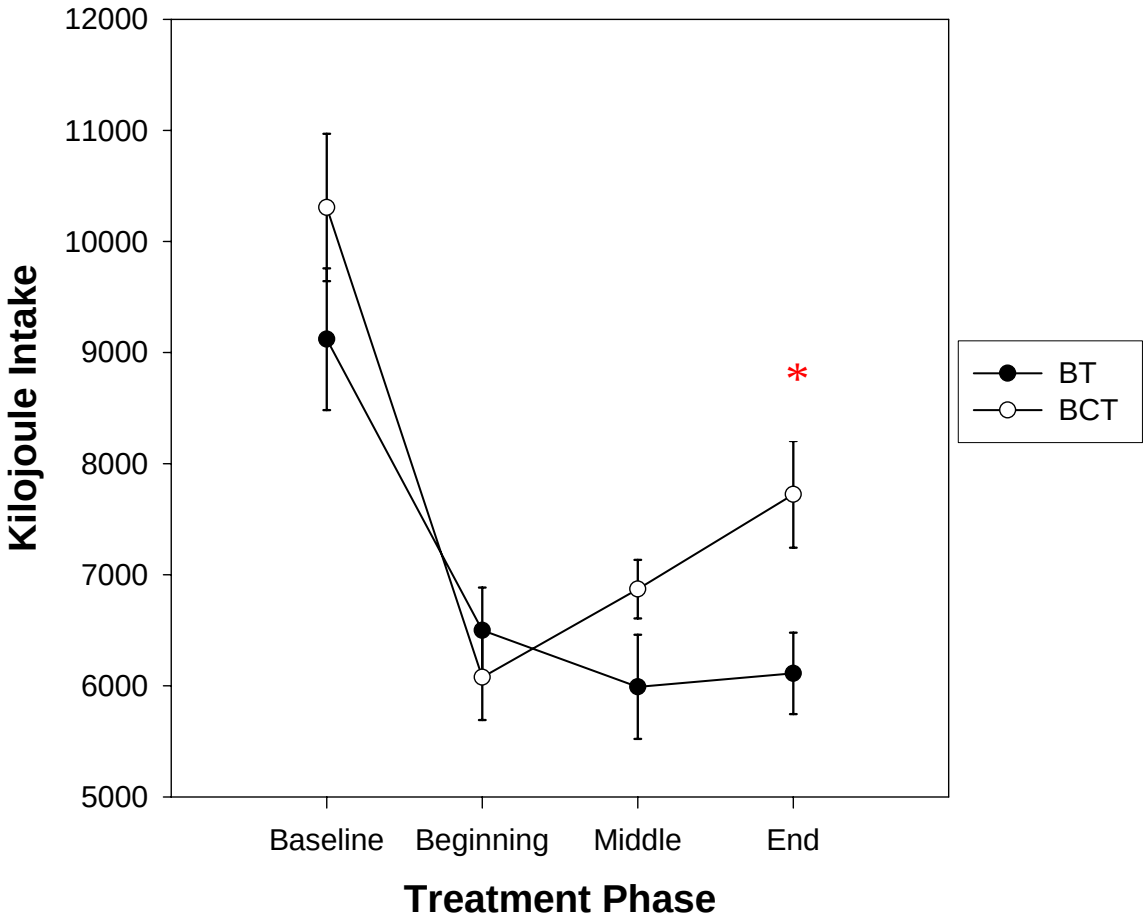


Figure 5. Mean Daily Kilojoule Intake Across Treatment

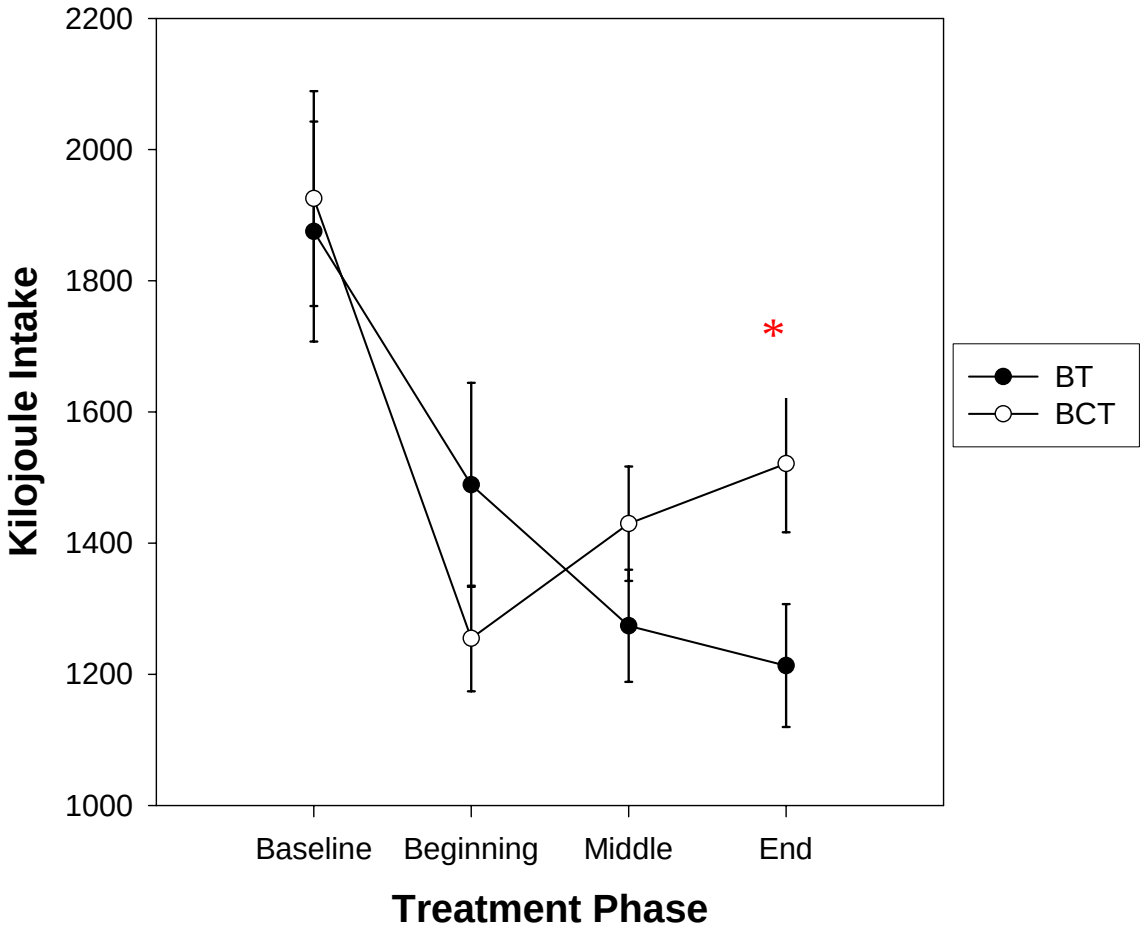


* Denotes statistical significance between groups differences at the $p < .05$ level

Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1, 15) = 18.7$, $p < .01$; BCT = $F(1, 14) = 35.6$, $p < .01$.

Within groups analyses revealed significant differences for BCT from the beginning to the end of treatment. BCT = $F(1, 14) = 5.89$, $p = 0.03$.

Figure 6. Mean Kilojoule Intake for Breakfast Across Treatment

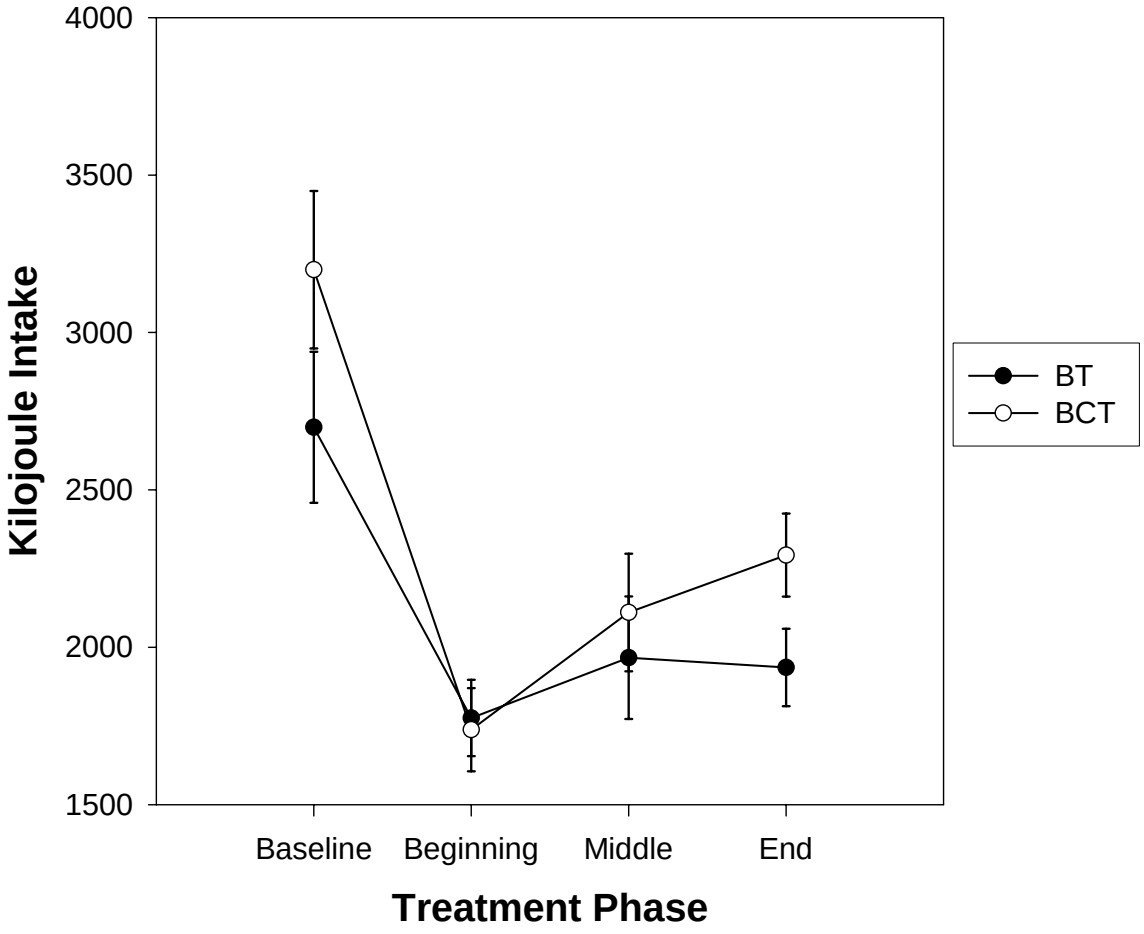


* Denotes statistical significance between groups at the $p < .05$ level

Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1, 15) = 4.56$, $p = 0.05$; BCT = $F(1, 14) = 17.8$, $p < 0.01$.

Within groups analyses revealed significant differences for BT and BCT from the beginning to the end of treatment. BT = $F(1, 15) = 4.28$, $p = 0.05$; BCT = $F(1, 14) = 5.48$, $p = 0.03$.

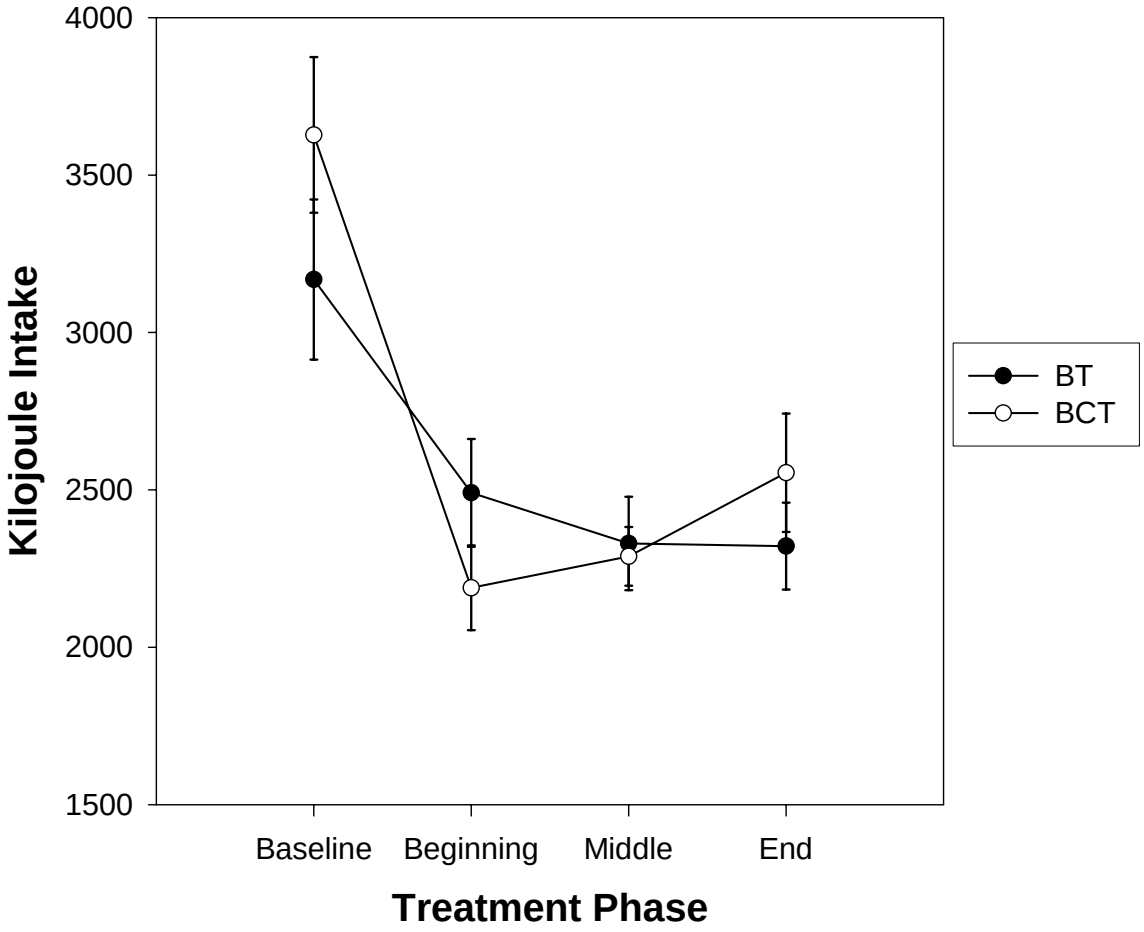
Figure 7. Mean Daily Kilojoule Intake for Lunch Across Treatment



Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1, 15)=17.59$, $p< 0.01$; BCT = $F(1, 14)=30.55$, $p< .01$.

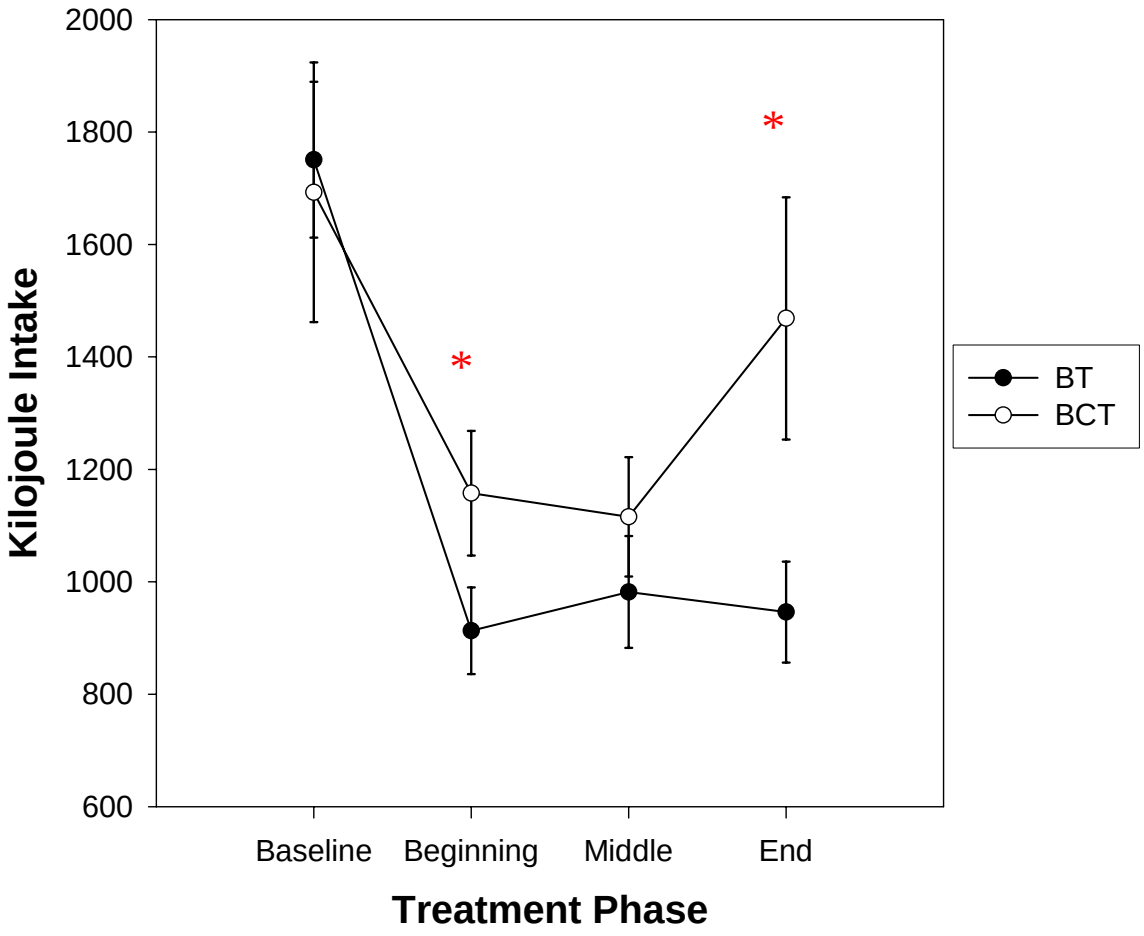
Within groups analyses revealed significant differences for BCT from the beginning to the end for treatment. BCT = $F(1, 14) =8.25$, $p= 0.01$.

Figure 8. Mean Dialy Kilojoule Intake for Dinner Across Treatment



Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = F(1, 15)=7.46, p= 0.02; BCT = F(1, 14)=36.9, p< 0.01.

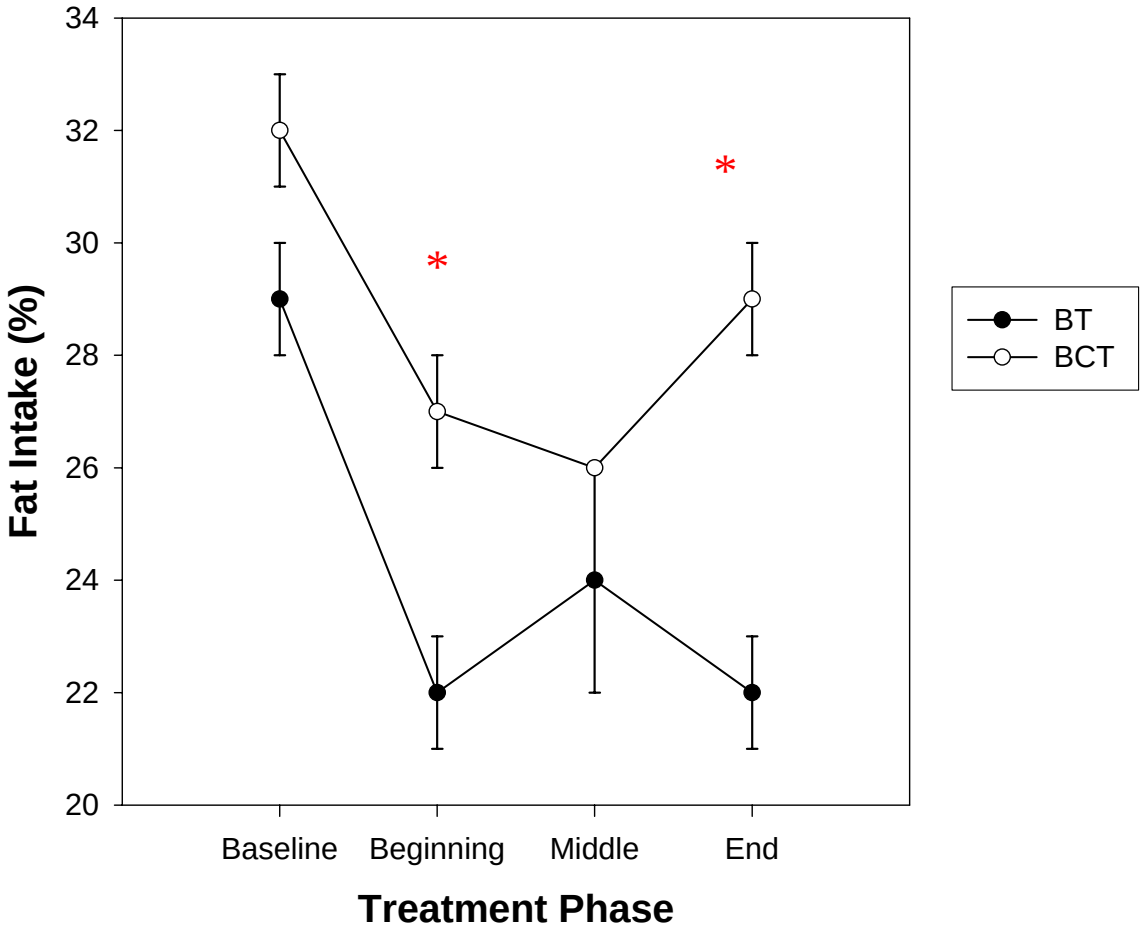
Figure 9. Mean Daily Kilojoule Intake for Snacks Across Treatment



* Denotes statistical significance between groups at the $p < .05$ level

Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1, 15) = 39.1$, $p < .01$; BCT = $F(1, 14) = 6.99$, $p = 0.02$.

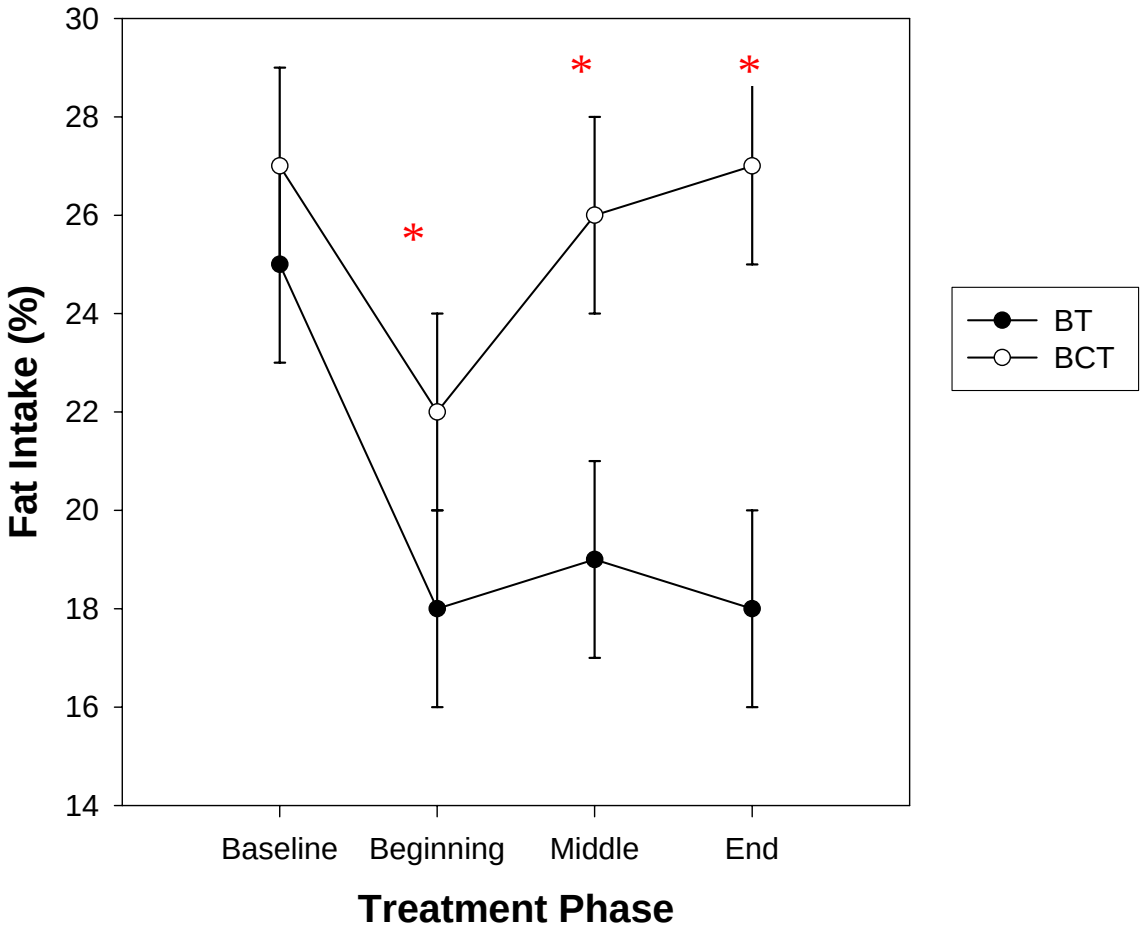
Figure 10. Mean Daily Percent Fat Intake Across Treatment



* Denotes statistical significance between groups at the $p < .05$ level

Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1, 15) = 21.09$, $p < .01$; BCT = $F(1, 15) = 10.73$, $p < .01$.

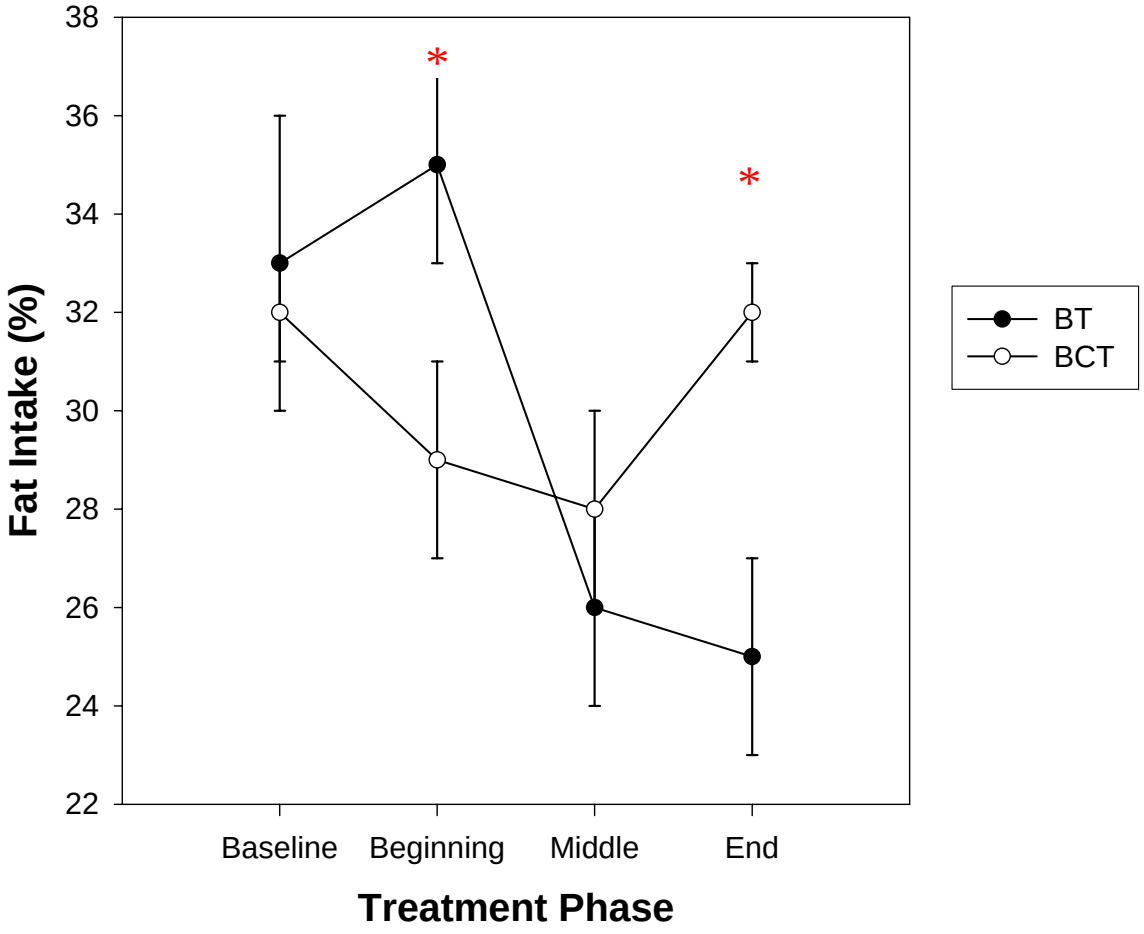
Figure 11. Mean Daily Percent Fat Intake at Breakfast Across Treatment



* Denotes statistical significance between groups at the $p < .05$ level

Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1, 15) = 12.3$, $p < .01$; BCT = $[F(1, 15) = 6.28$, $p = 0.02$.

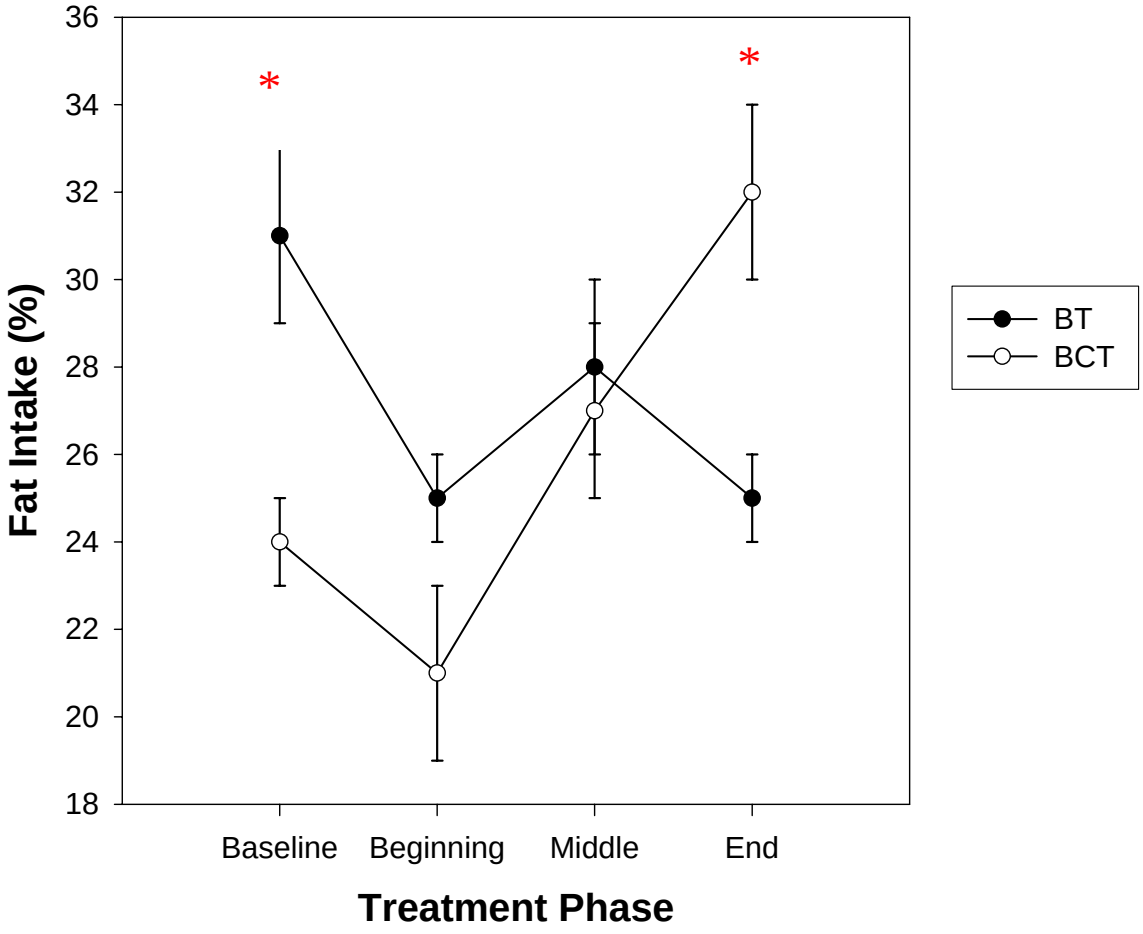
Figure 12. Mean Daily Percent Fat Intake at Lunch Across Treatment



* Denotes statistical significance between groups at the $p < .05$ level

Within group analyses revealed significant differences from baseline to the beginning of treatment for BT. BT = F(1, 15)=7.27, p= 0.02.

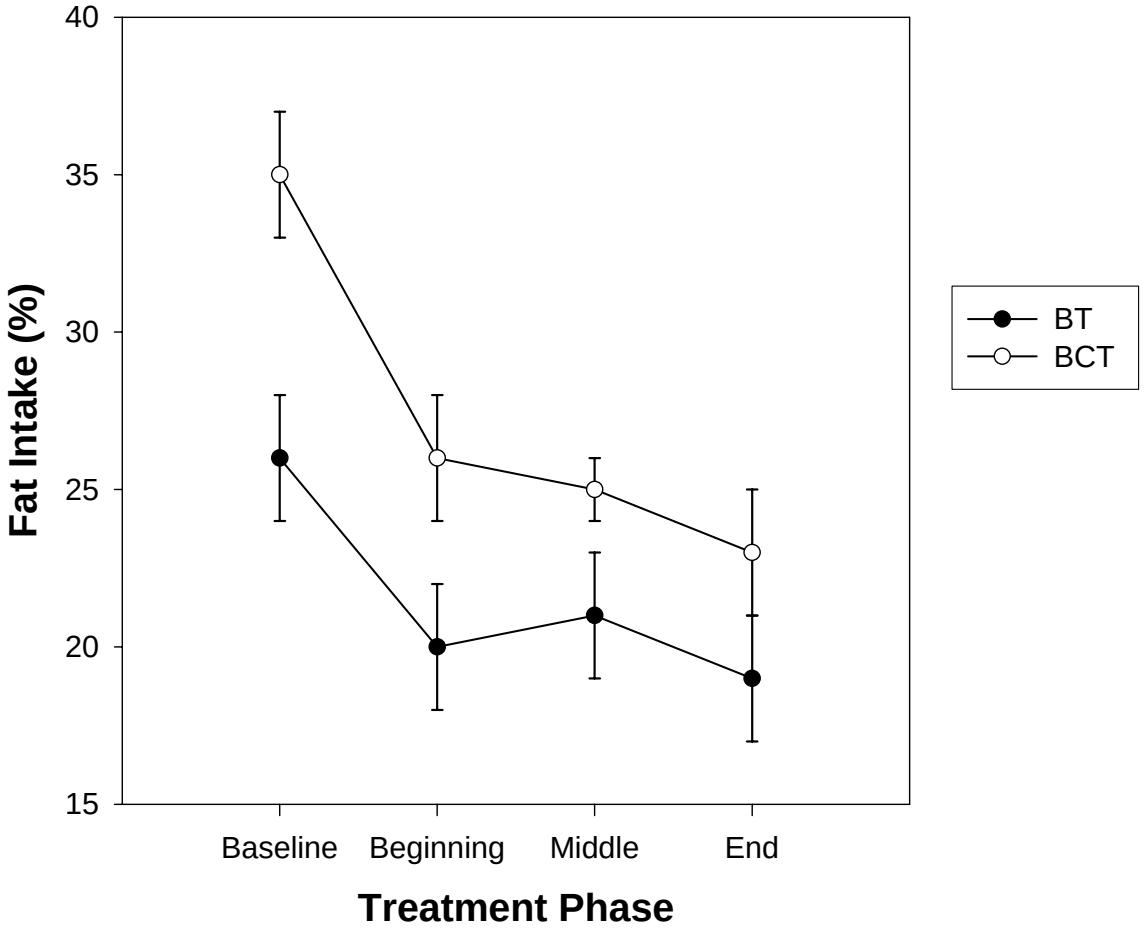
Figure 13. Mean Daily Percent Fat Intake at Dinner Across Treatment



* Denotes statistical significance between groups at the $p < .05$ level

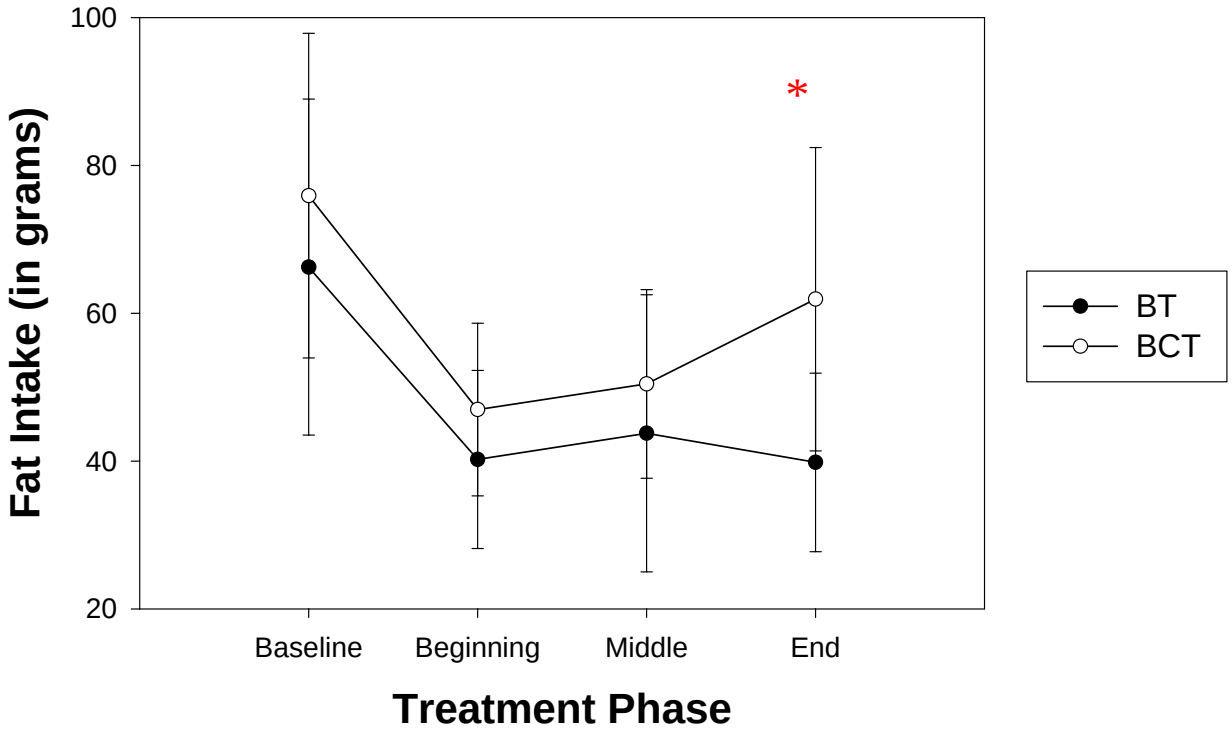
Within group analyses revealed significant differences from baseline to the beginning of treatment for BT. BT = $F(1, 15) = 12.73$, $p < .01$.

Figure 14. Mean Daily Percent Fat Intake at Snacks Across Treatment



Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1,15)=7.95$, $p= .01$; BCT = $F(1, 15)=12.43$, $p< .01$.

Figure 15. Mean Daily Fat Gram Intake Across Treatment

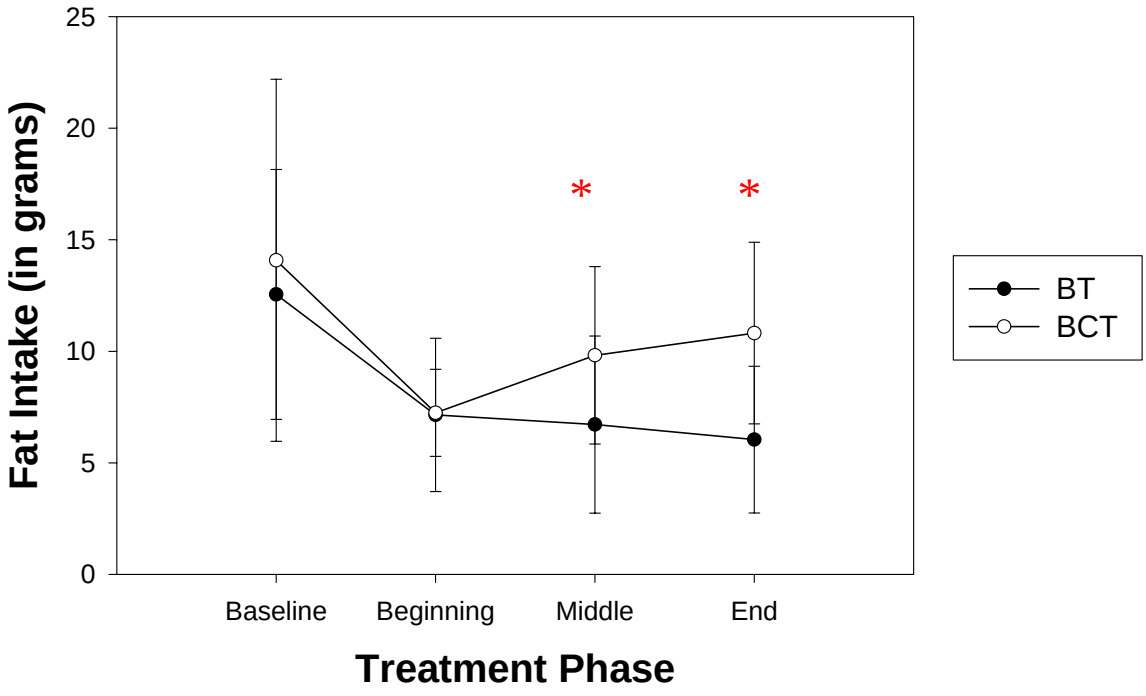


* Denotes statistical significance between groups at the $p < .05$ level

Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1, 15) = 38.67$, $p < .01$; BCT = $F(1,14) = 39.46$, $p < .01$.

Within groups analyses revealed significant differences for BCT from the beginning to the end for treatment. BCT = $F(1, 14) = 6.58$, $p = 0.02$.

Figure 16. Mean Daily Fat Gram Intake at Breakfast Across Treatment

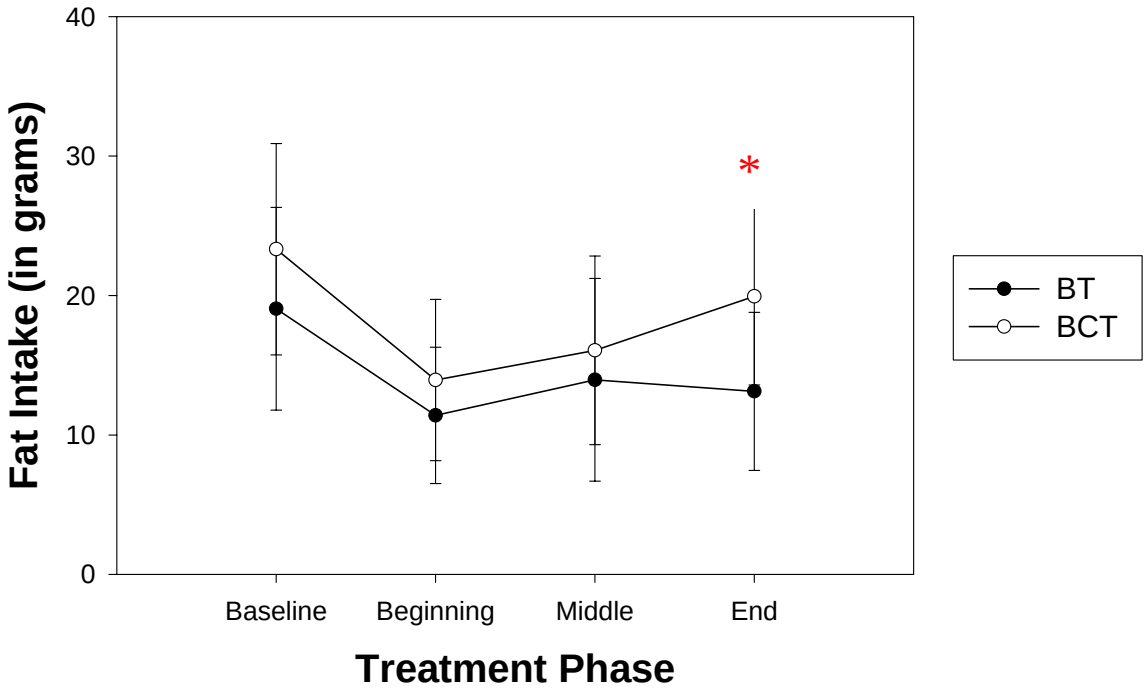


* Denotes statistical significance between groups at the $p < .05$ level

Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1, 15) = 20.37$, $p < 0.01$; BCT = $F(1, 14) = 11.93$, $p < 0.01$.

Within groups analyses revealed significant differences for BCT from the beginning to the end for treatment. BCT = $F(1, 14) = 16.19$, $p < 0.01$.

Figure 17. Mean Daily Fat Gram Intake at Lunch Across Treatment

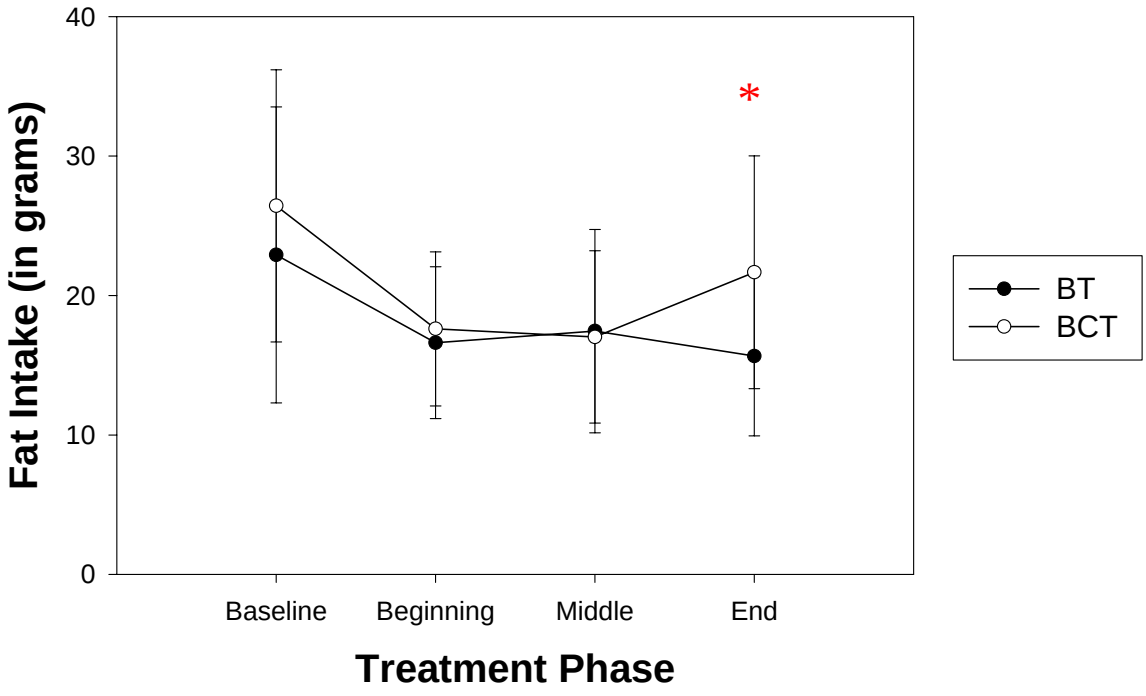


* Denotes statistical significance between groups at the $p < .05$ level

Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1, 15) = 21.43$, $p < 0.01$; BCT = $F(1, 14) = 20.98$, $p < 0.01$.

Within groups analyses revealed significant differences for BCT from the beginning to the end for treatment. BCT = $F(1, 14) = 6.06$, $p = 0.03$.

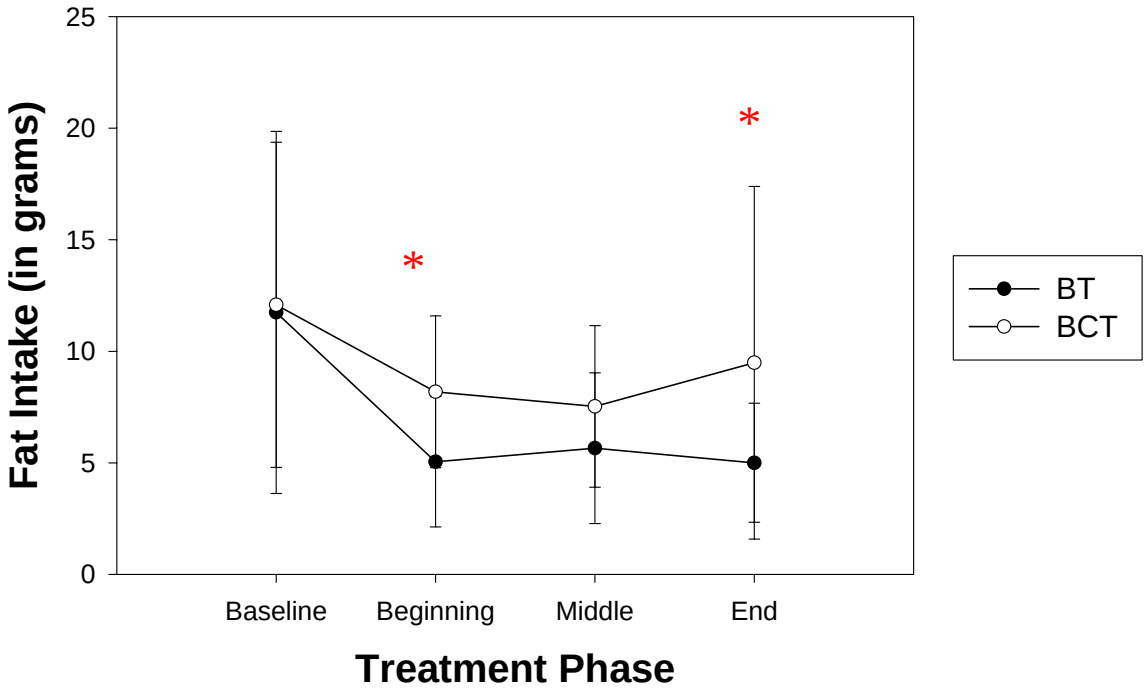
Figure 18. Mean Daily Fat Gram Intake at Dinner Across Treatment



* Denotes statistical significance between groups at the $p < .05$ level

Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1, 15) = 5.98$, $p = 0.03$; BCT $F(1,14) = 16.77$, $p < 0.01$.

Figure 19. Mean Daily Fat Gram Intake at Snacks Across Treatment

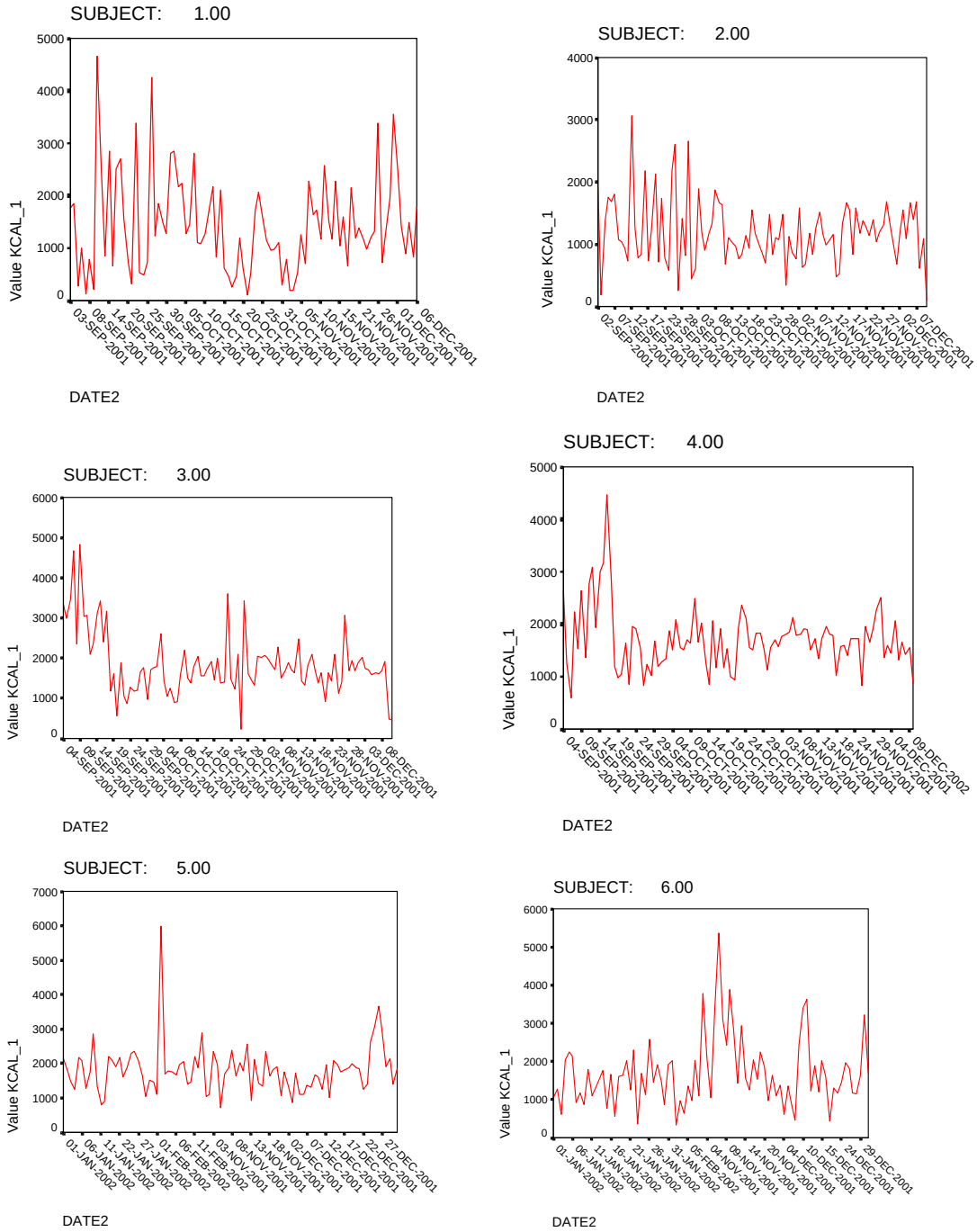


* Denotes statistical significance between groups at the $p < .05$ level

Within group analyses revealed significant differences from baseline to the beginning of treatment for both BT and BCT. BT = $F(1,15)=13.18$, $p< 0.02$; BCT $F(1, 15)=4.95$, $p= 0.04$.

Figure 20. Mean Daily Variability in Kilojoule Intake by Participant and by Group

BEHAVIOR THERAPY GROUP: Participants 1-16



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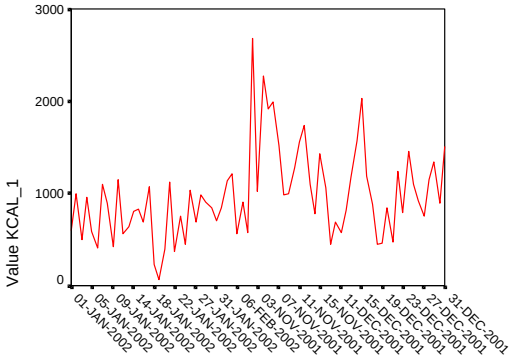

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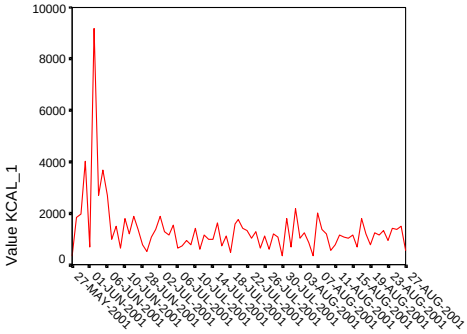

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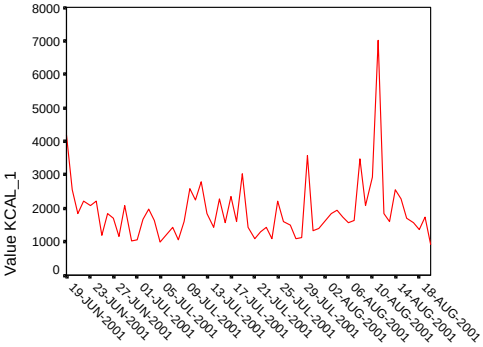

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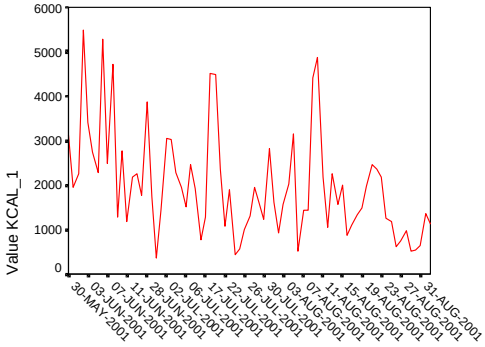

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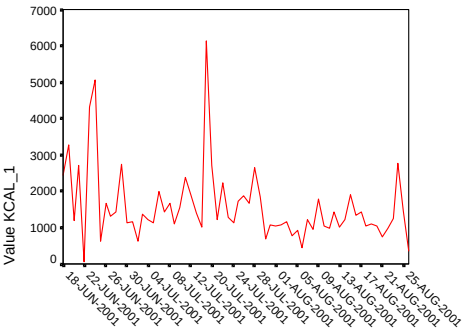

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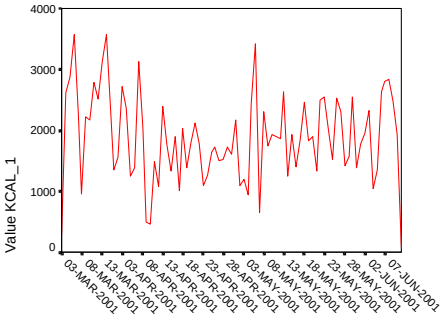

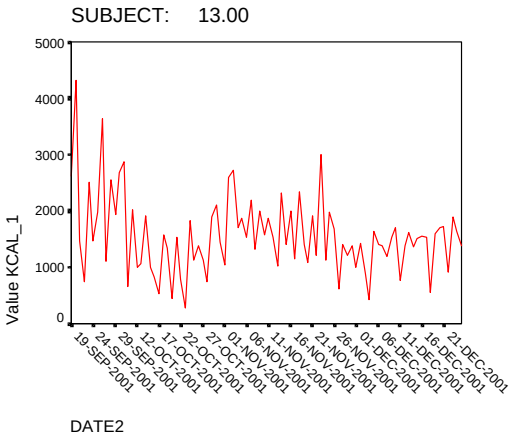
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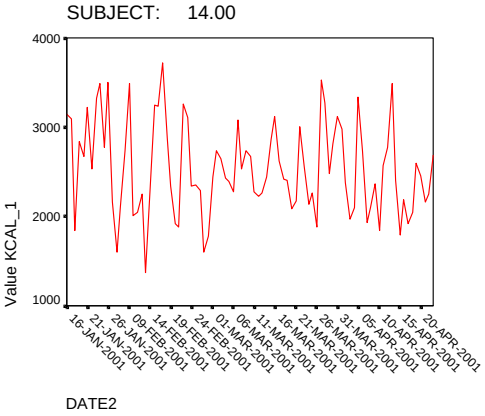
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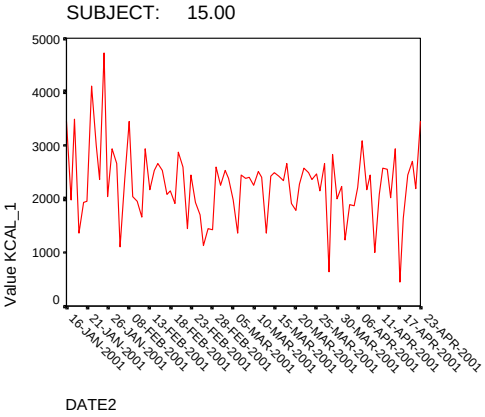
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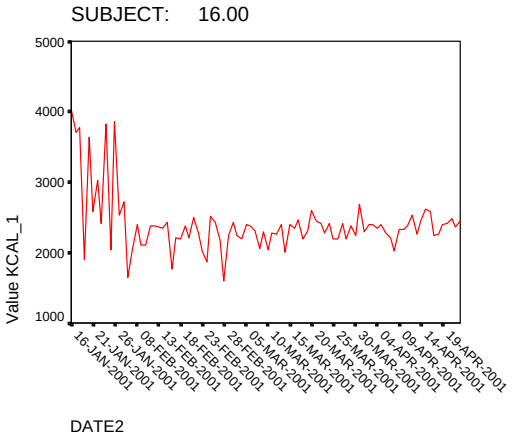
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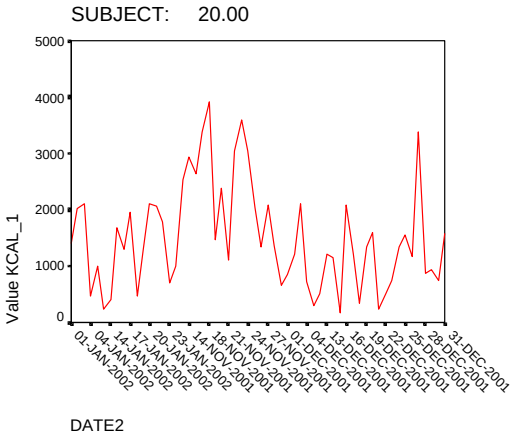
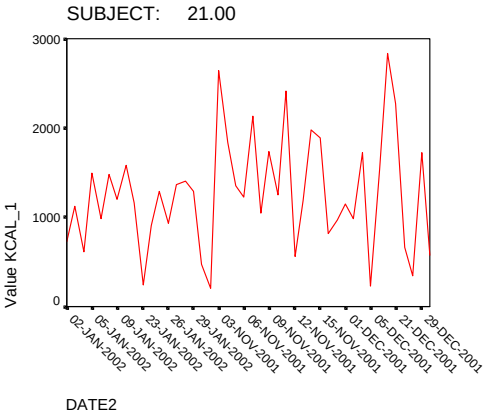
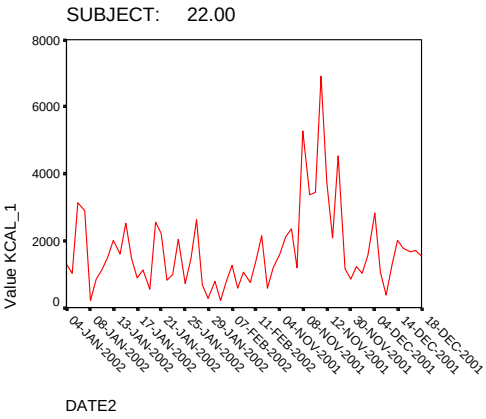
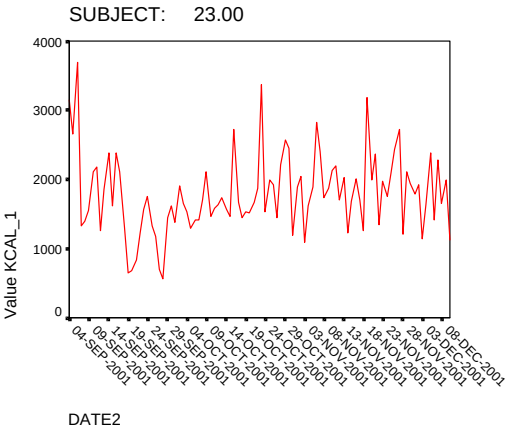
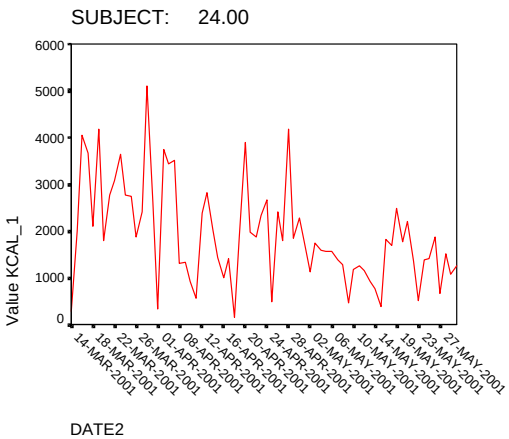
BEHAVIOR THERAPY GROUP: Participants 20 – 35

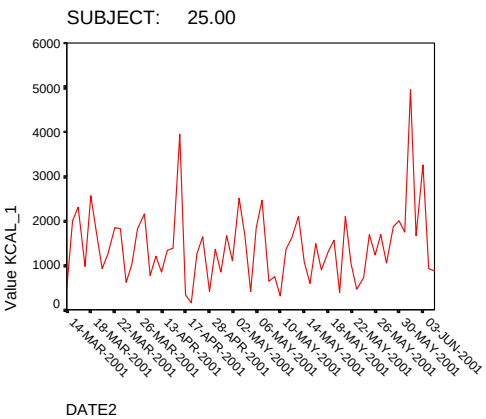


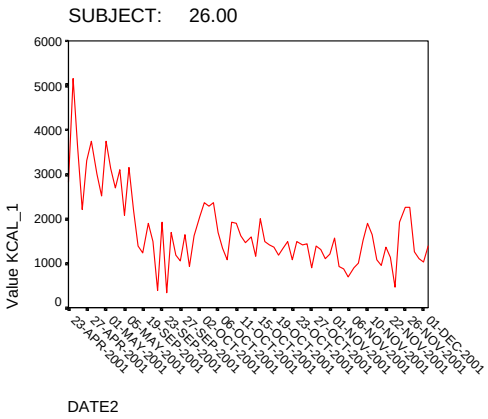








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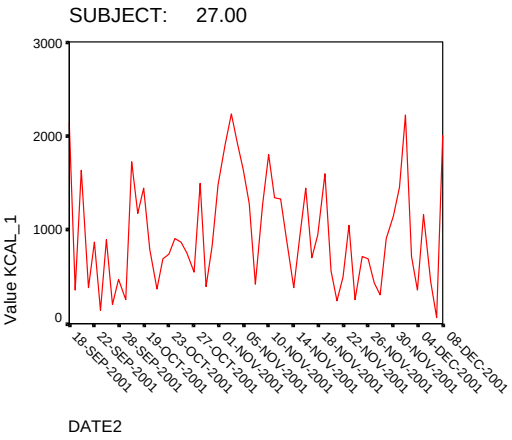
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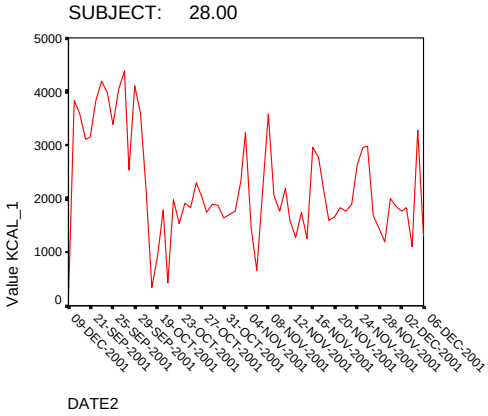
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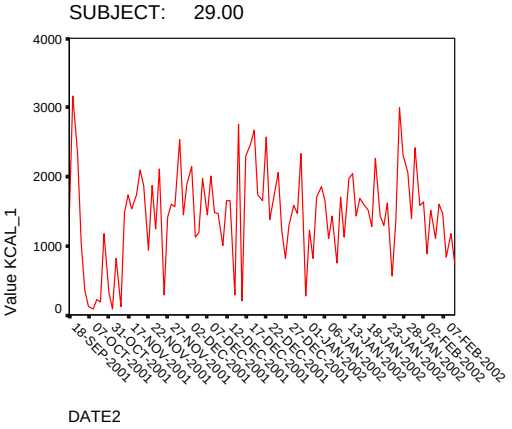
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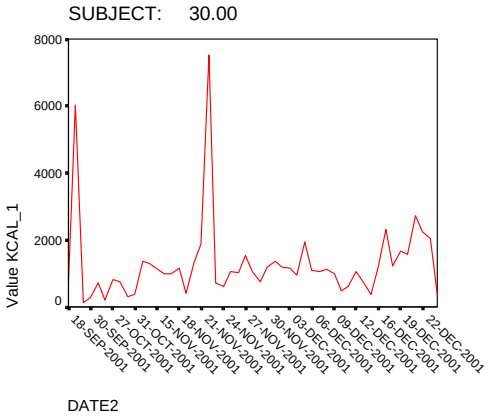
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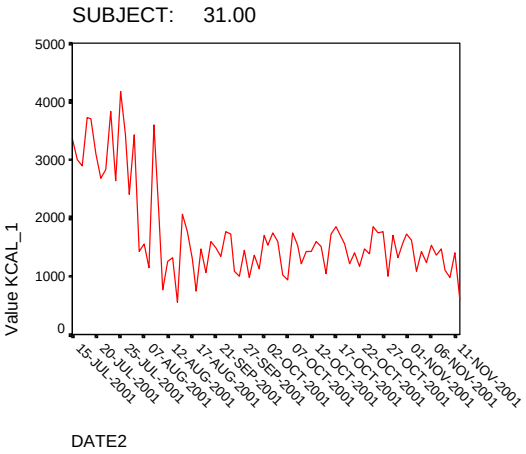
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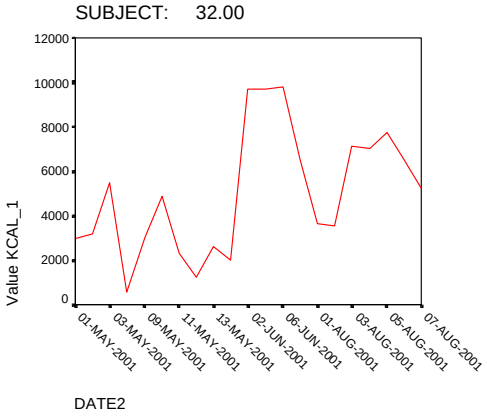
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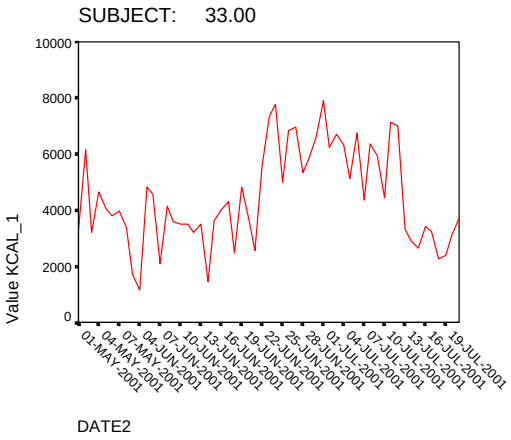
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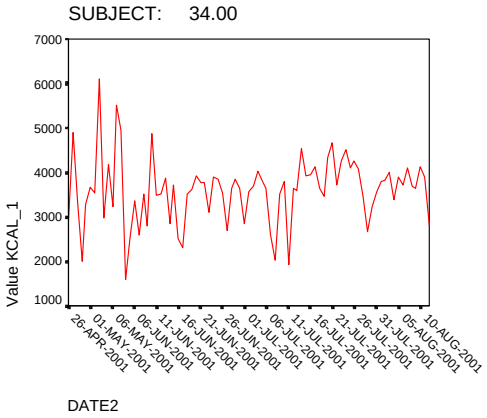
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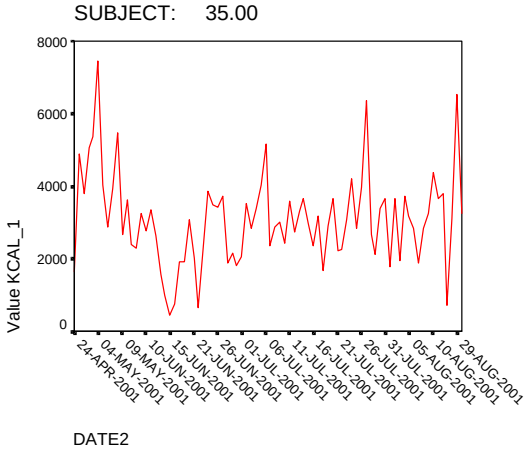
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Figure 21. Changes in Mean Daily Standard Deviation of Kilojoule Intake by Group Across Treatment

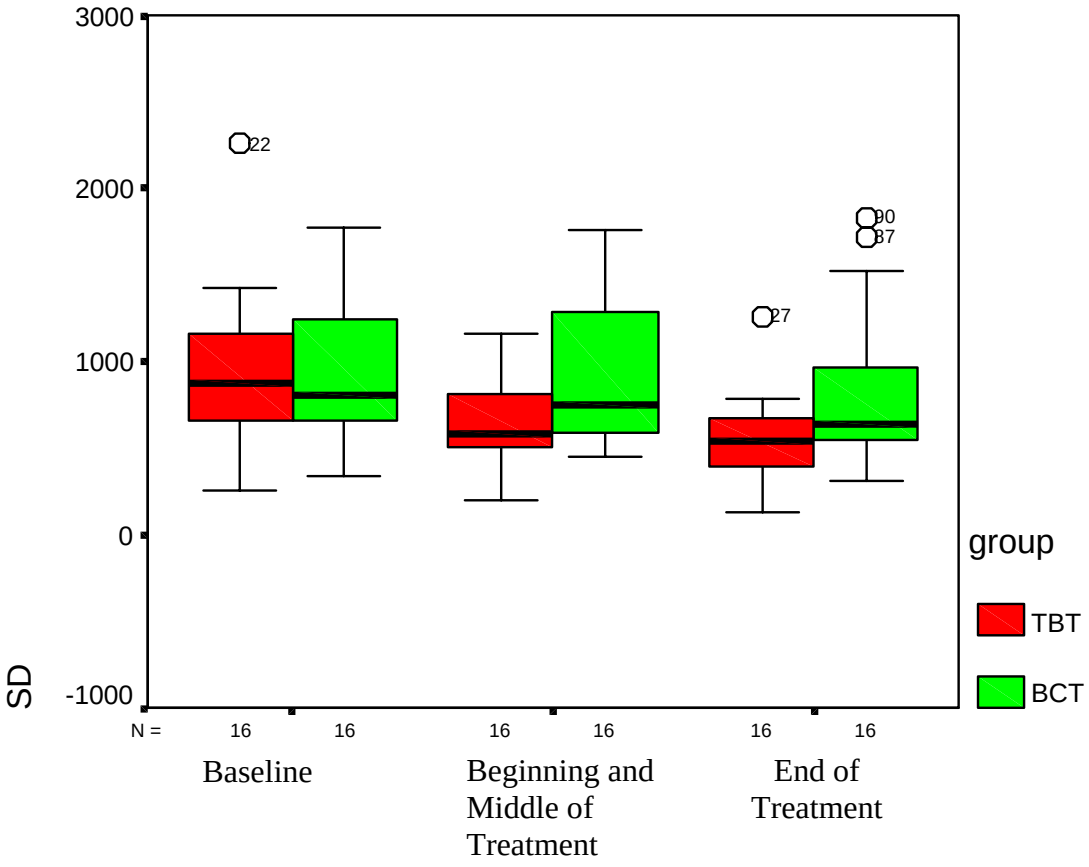

Figure 22. Mean Daily Number of Eating Episodes Across Treatment

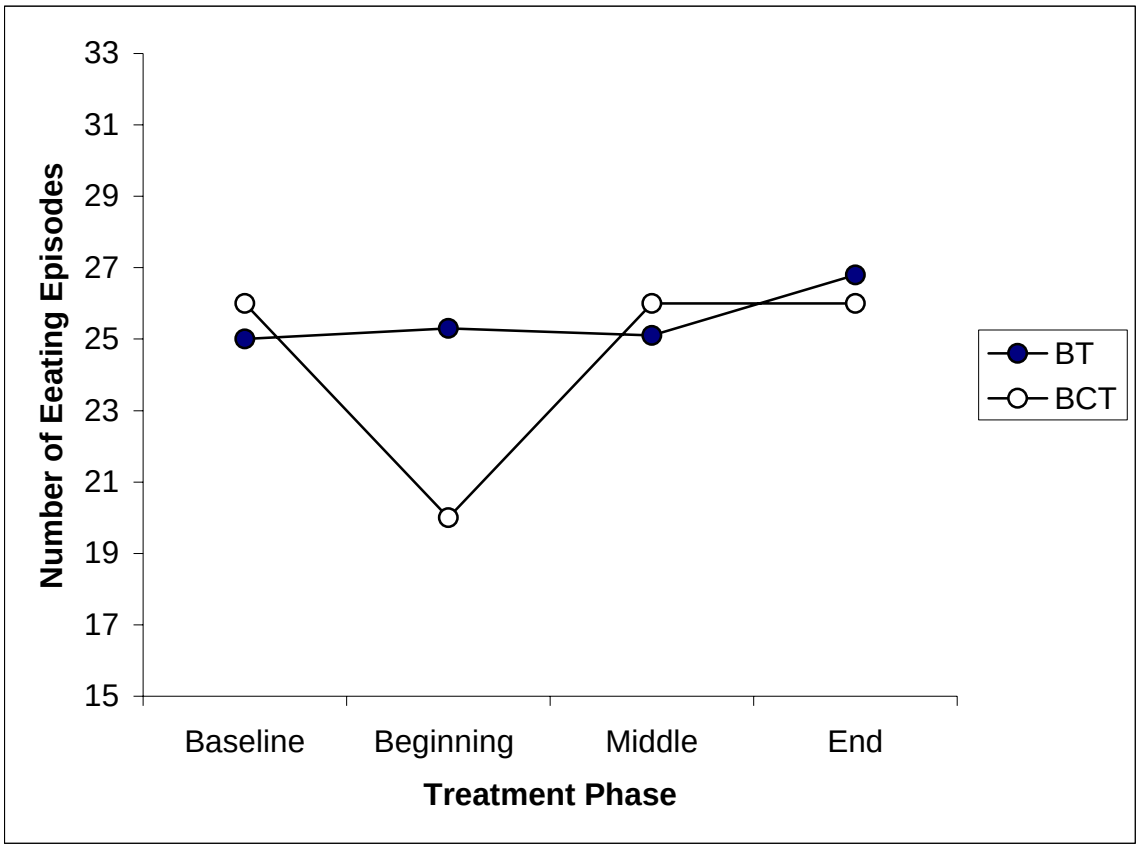

Figure 23. Mean Daily Number of Eating Episodes for Breakfast Across Treatment

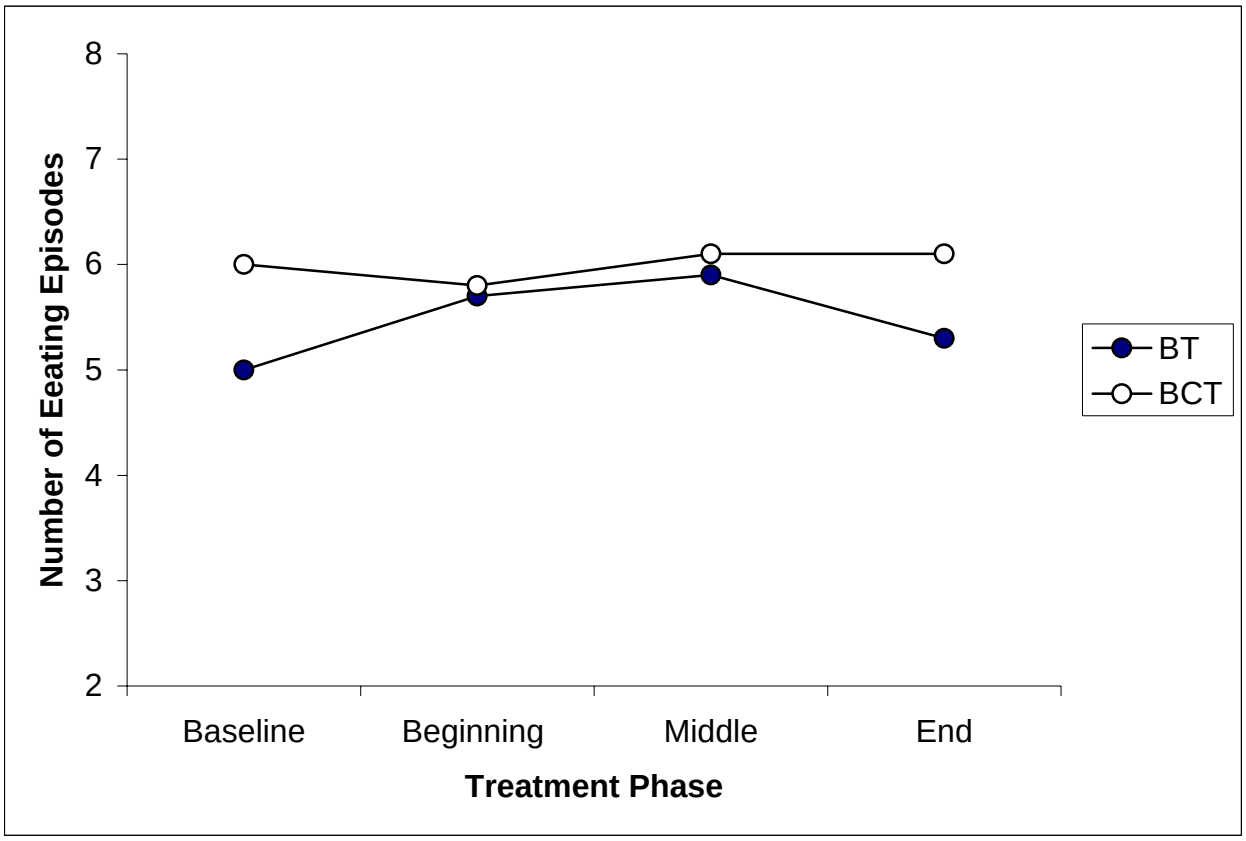

Figure 24. Mean Daily Number of Eating Episodes for Lunch Across Treatment

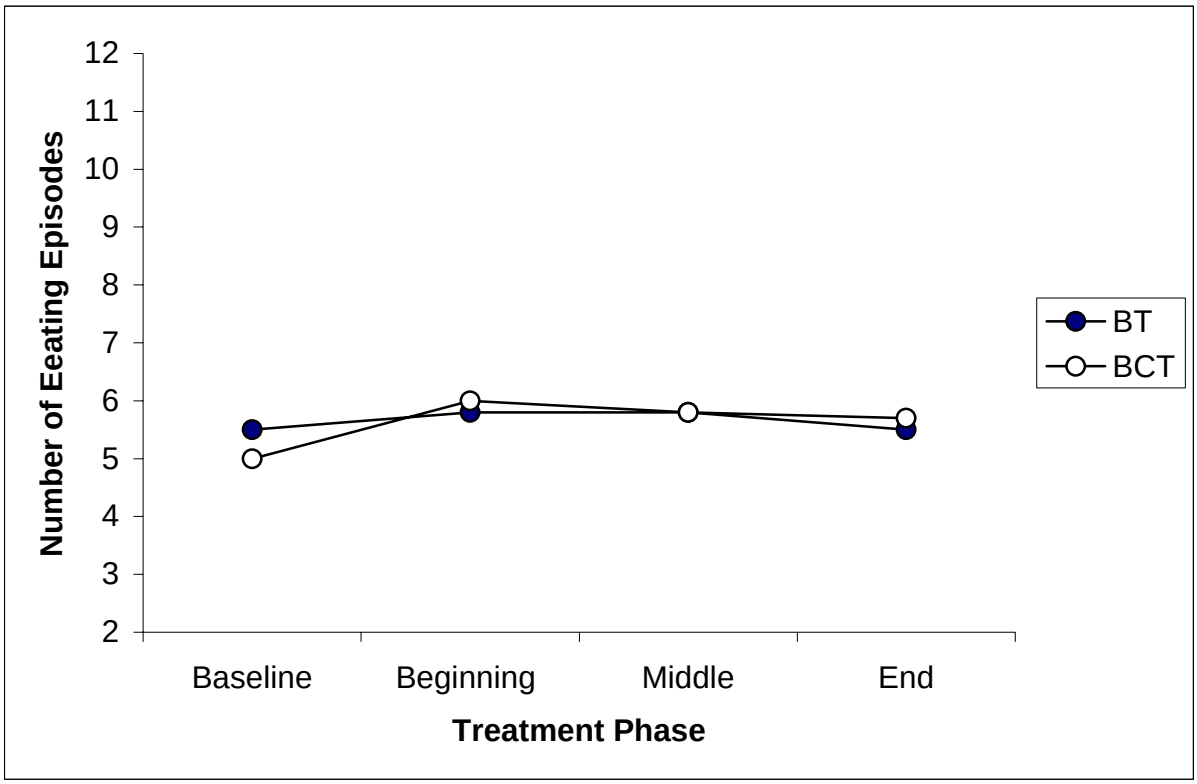

Figure 25. Mean Daily Number of Eating Episodes for Dinner Across Treatment

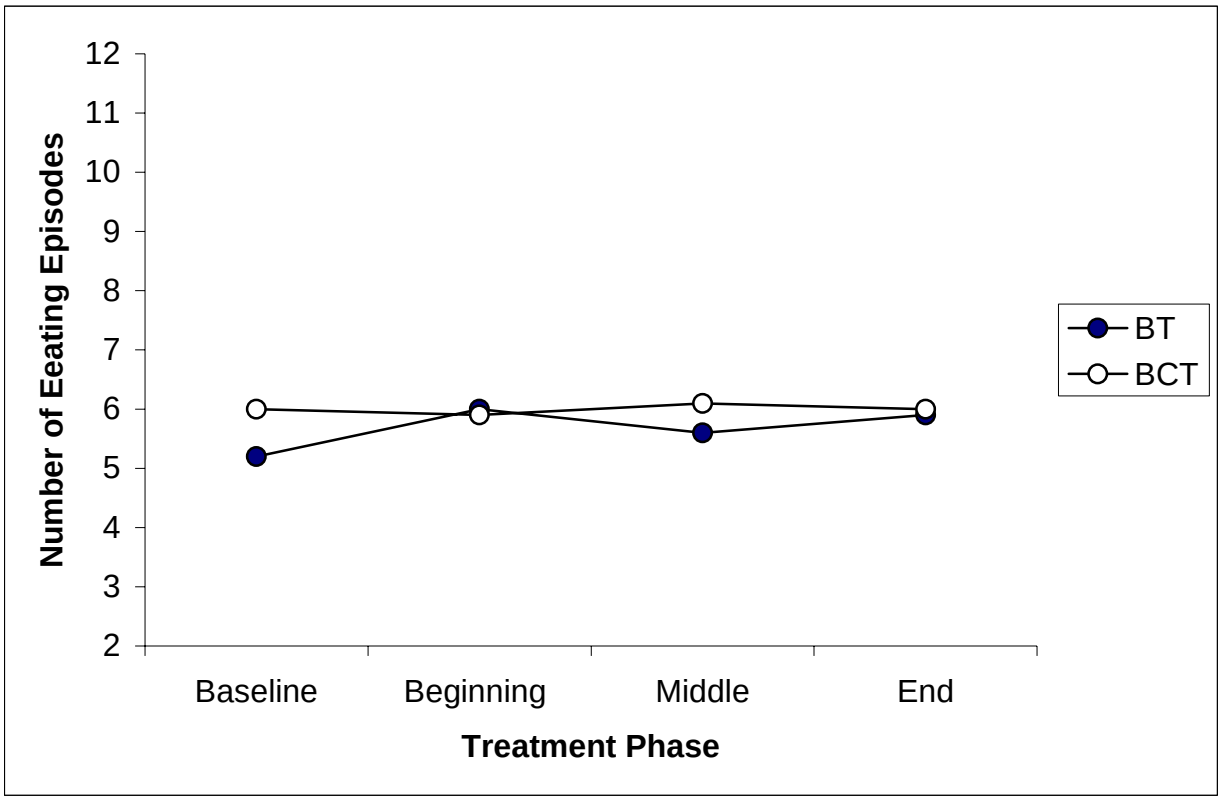

Figure 26. Mean Daily Number of Eating Episodes for Snacks Across Treatment